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THE
LARYNGOSCOPE.

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RHINOLOGY IN CHILDREN.
RESUME OF THE LITERATURE FOR 1954.

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This resumé of the literature on Rhinology in Children comes from the Hospital for Sick Children in Toronto, and the Department of Otolaryngology of the University of Toronto. The medical journals, including the pediatric publications of the United States, Britain and Canada, have been reviewed. The classification of previous years has been maintained.

GENERAL ARTICLES.

Dodwell[1] in the latest edition (the sixteenth) of *Principles and Practice of Medicine,* orginally edited by Osler, defines status lymphaticus as "a combination of constitutional anomalies, among which are hyperplasia of the lymphoid tissues and of the thymus gland, hypoplasia of the cardiovascular system and peculiarities of configuration, with frequent sudden death".

Results of the condition are various; among them are: *1.* Liability to sudden death—this may be from anaphylaxis, cerebral hemorrhage or, in young children, pressure on the trachea by the enlarged thymus gland; *2.* Increased suscepti-

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bility and decreased resistance to acute infections; *3*. Increased danger in childbirth; and *4*. Psychical instability—these subjects forming a large proportion of cases of drug addiction and suicides.

The most circumstantial account of the condition is given by Hewer (1953). According to him the findings of Kemp (1932), Campbell (1937), Moncrieff (1938), and the late Sir Bernard Spilsbury, in spite of several attempts to discredit their work, have definitely established the existence of the syndrome. It would seem that Hewer bases his case that status lymphaticus exists as a clinical entity upon three claims:

1. That the condition has been recognized for some 300 years.

2. That there is a recognized clinical picture with three classical signs.

3. That there are well-recognized post-mortem findings.

Dodwell proposes to show that not one of these claims is supported by the available evidence.

If Dr. Hewer's story were correct we should expect to be able to follow these references to status lymphaticus back to the earliest editions of current works, and further back still in the earlier editions of books now out of print but in existence before Osler's *Medicine* was produced.

Actually there is no reference to the condition in Osler's first and second editions, nor in any contemporary medical or pathological book Dodwell has been able to discover. In fact it is not until the third edition of Osler's *Medicine*, published in 1898, that any reference is made, and then in these terms: "Much attention has been paid lately to a somewhat rare condition . . .," and the reader is advised to consult an article by Ewing (1897).

Ewing in discussing the views of Paltauf (1889) begins: "There have recently been described a series of cases . . . ". He continues: "The importance of the constitutio lymphatica and its relation to some forms of sudden death, especially to fatalities under chloroform narcosis, have been recognized

by the Vienna school of pathologists, but have received very scant attention elsewhere".

Six successive editions of Osler's *Medicine* carried the passage based upon Ewing's article but without his final sentence, which ran: "In conclusion, while the studies of the Vienna observers seem to have placed the existence of the constitutio lymphatica upon a firm basis in pathology, the observations yet remain far too limited fully to demonstrate the truth of an hypothesis which connects a large class of sudden deaths with simple hyperplasia of the lymphatic structures of the body".

It is surely significant that Paltauf, in a long list of references, makes no mention of any work published earlier than 1850; equally so that John Simon (1845)—although his bibliography is immense, going back as far as Galen and Rufus of Ephesus—seems entirely to have missed this historical association.

The history of the growth of the legend of status lymphaticus can be quickly told. Gradually during the first decade of the twentieth century the idea of a status lymphaticus began to spread, so that in the eighth and ninth editions of Taylor's *Practice of Medicine*, in 1908 and 1911, references begin to appear, and in 1911 for the first time a separate entry of diseases of the thymus gland makes its appearance in the annual report of the Registrar-General for England and Wales.

In 1917 Dr. Symmers, professor of pathology at the Bellevue Hospital, New York, published an original work upon status lymphaticus in which he recorded the weights of the thymus gland in 66 cases out of 249 which he had diagnosed "status lymphaticus".

The weakness of Dr. Symmer's case is that he publishes no standard for normal weights of the thymus gland, and if we compare the weights of his enlarged glands with the normal weights arrived at by Hammar (1916), or by the Medical Research Council's Joint Committee (1931), whose findings were based on a much larger published collection, we find that a preponderance of these abnormally heavy glands of Dr.

Symmer's fall within these other observers' normal weights; however, Dr. Symmer's views have gained much popularity and have formed the basis of the description of the condition in Osler's *Medicine* from the eighth edition, of 1920, to the latest, (the sixteenth), published in 1947.

The legend of the 300-year-old tradition was not launched in British medical literature until the appearance of the second edition of Hewer's *Recent Advances in Anesthesia and Analgesia*, in which an article by Platter is quoted. The latter died in 1614 a few months after the publication of his work *Observationum in Hominis Affectibus*, a reprint of which is to be found in the library of the Royal Society of Medicine.

Platter's name was brought to the attention of American medical men in 1912 by C. G. Cumston, but his name does not appear in connection with status lymphaticus until 1924, when Ruhrah commented: "To pediatrics we know he made one contribution of great value, the description of thymus death".

With regard to the recognized clinical picture, the three classical signs are mentioned for the first time by Kemp (1932), who quotes no case notes to support his statements. No other writer records cases presenting these physical signs. Campbell (1937) lists an impressive number of physical signs, far too numerous to be mentioned here, associated with status lymphaticus but, like Kemp, fails to favor us with any case records.

That there are well recognized post-mortem findings is not supported by text books of pathology, although the writers of such works must have much greater experience of the post-mortem findings in such cases.

Thus Boyd (1947) states that "there is no proof that the thymus gland has anything to do with stoppage of the heart". Dible and Davie (1950) state: "There is no doubt that ignorance of the great variation in size to which the thymus gland is subject in health and disease has often led to an unjustifiable resort to the convenient diagnosis of status lymphaticus in cases of sudden death . . . Many pathologists of experience are of the opinion that there does exist some abnormal con-

stitution or condition which is associated . . . with a liability to sudden death from relatively trivial stimuli, such as excessive sensitivity to stimuli of the autonomic nervous system, but no constant anatomical or pathological abnormality".

Muir (1951) quotes the findings of the Joint Committee of the Medical Research Council and of the Pathological Society of Great Britian and Ireland, its general conclusions being that the facts that have been ascertained afford no evidence that the so-called status thymo-lymphaticus has any existence as a pathological entity.

The conclusion in Muir's textbook is that "this does not imply that there is not a constitutional condition of low resistance power, with a tendency to sudden death; simply that it is not possible to relate it definitely to thymic or lymphoid hyperplasia".

As to Sir Bernard Spilsbury's post-mortem findings in status lymphaticus, the authors have been completely unsuccessful in their attempt to discover any published work in which such findings are quoted. In 1926 there was set up a Joint Committee of the Medical Research Council and of the Pathological Society of Great Britian and Ireland, which published its report in 1931. Some 680 post-mortem examinations were made, and the care and detail with which these cases were analyzed place this investigation in a class by itself.

The diagnosis of enlarged thymus glands by X-ray is a very unreliable basis, as the writings of Garland (1929) and of Capper and Schless (1934) have emphatically shown.

The author suggests that were this account of status lymphaticus other than a fable, ample material in support could have been found among the thousands of post-mortem examinations reported since 1945 by Helpern and Rabson (1945), Keith Simpson (1947), Richards (1947), Deadman (1947) and others, or from among the 200 odd deaths reported annually by the Registrar-General for England and Wales.

In conclusion the authors believe that the 12 conclusions of the Joint Committee of the Medical Research Council and of the Pathological Society of Great Britain and Ireland re-

main the considered judgment of informed opinion, and that the last sentence of their report holds good: "In the opinion of the Joint Committee the facts elicited in the present inquiry are in harmony with those of Hammar (1926, 1929), and of Greenwood and Woods (1927) in affording no evidence that so-called status thymo-lymphaticus has any existence as a pathological entity".

Because periorbital edema is often associated with acute ethmoiditis in children we present here a case report by Bass[2] of periorbital edema as the initial sign of infectious mononucleosis. This 12-year-old girl came home from school feeling tired and with a temperature of 104.8°F. The most striking feature on examination was the condition of her eyelids and periorbital tissues. The eyelids were definitely puffy but not reddened. There was no tenderness over the antra or ethmoid sinuses. The throat and tonsils were not inflamed. The urine was normal on examination. Because of these findings and a negative allergic history, infectious mononucleosis was suspected. There were no lymph nodes and the spleen was not palpable, but a blood smear showed the typical findings of infectious mononucleosis. The cause of the lid edema is obscure, but may be due to lymphatic obstruction, even if no peripheral lymph nodes were palpable.

Evans[3] in a long article with ample bibliography reviews the treatment of epistaxis which although commonly easy to correct may at times constitute a difficult surgical emergency.

He states that the incidence is highest in children but seldom serious. Often the simple nose bleed will stop spontaneously regardless of treatment used, or even without treatment. Although this is true, it must also be remembered that to the patient this fact may not be known or obvious. Whenever the bleeding from the nose is of sufficient severity that a physician is consulted, it is to the patient, or to his family, a serious matter and sometimes a frightening occurrence.

For this reason, reassurance is the first step in treatment. If the patient is at home, instruction should be given that he should be placed in the proper position, that is, that he should sit up and bend forward. If he lies on his back, the stomach

may fill with blood, causing faintness and nausea. In cases of simple nose bleed from the anterior septum, blowing out the clots and pressing the anterior nares together for a few minutes may be sufficient to control the condition.

Whenever intranasal manipulation or packing is needed, the administration of a sedative is indicated, whether the patient be a child or an adult. Once his anxiety is relieved and he becomes relaxed, whatever measures are necessary to control the bleeding are much more easily accomplished. In cases of anterior nasal bleeding from Kiesselbach's or Little's area, the bleeding vessel can frequently be seen, if the hemorrhage is not too profuse; then the area is cocainized and cauterized either with one of the chemical agents or the electric current.

In attempting to control hemorrhage from the anterior part of the nose of a child, his associates and he prefer the use of chemical cautery to electrocoagulation. In many instances, they use a solution of 50 per cent silver nitrate, which is immediately neutralized by sodium chloride solution and covered with holocaine and adrenalin ointment. Some authors suggest the use of a silver nitrate stick or chromic acid or trichloracetic acid; the latter two should be neutralized by a solution of sodium bicarbonate.

When the hemorrhage in the anterior portion of the nose is so severe that it is impossible to find the bleeding point, the nose is packed with cotton pledgets saturated with cocaine and adrenalin until the hemorrhage ceases. Then an attempt is made to find the bleeding point in order that the area may be cauterized.

When the hemorrhage is from the anterior superior part of the nose, and especially when there is a history of recurrent or profuse bleeding, he has found it most satisfactory to use "spot" packing of oxidized cellulose or gel foam saturated with thrombin and held in place with a cotton sponge and a small piece of adhesive placed over the nostril to prevent the pack from being dislodged by sneezing. The cotton should be saturated with penicillin solution to reduce the chance of infection. He also administers an antibiotic systemically if the pack is to be left in place as long as 24 hours.

In case of recurrent nasal bleeding in children, which the parents usually say is spontaneous and frequently comes on at night, there is often a discharge with crusting of the septum near the anterior nares which may be due to infection or to allergy. The child with allergic rhinitis has a tendency to rub the nose to relieve the itching sensation and to pick at the crusts with the finger. Excessive drying of the nasal mucus greatly reduces its protective value and renders the capillary bed more accessible to trauma.

A measure devised by Hilding and recommended by Hunnicutt is helpful in preventing crusting and allowing the tissues to return to normal; this is to place a light plug of dry cotton in the nostril for a few minutes several times a day. In cases in which any nasal discharge is present, it is important that appropriate treatment for the underlying condition be instituted.

In all instances of continued or recurrent epistaxis in children, a thorough clinical examination and appropriate laboratory studies are indicated, to determine whether the bleeding is being caused by some systemic disease. Nasal hemorrhage of systemic origin in children is frequently noted in diseases of the blood, liver or kidneys, and in acute rheumatic fever. In such instances, treatment of the underlying disease is of paramount importance, with the local measures which are necessary for the control of bleeding from the nose.

SINUSITIS.

Asked "to what extent does bone regeneration occur in the lateral wall of the nose after antrostomy", the British Medical Journal[4] replied as follows.

"After antrostomy, a certain amount of new bone formation occurs around the edges of the opening made between the antrum and the inferior meatus of the nose, and if small fragments of bone are left adherent to the mucous membrane of either nose or antrum the new bone formation is more likely to occur. This is more likely to occur in children and where there is persistent infection. The bone formation causes progressive narrowing of the opening until it may become completely closed, and the thin layer of bone separating the two

cavities may be replaced by a thicker and tougher one. There is, however, no encroachment on the cavity of the nose or antrum".

Marrañon and Armengaud[5] examined the radiographic images of 365 normal children of both sexes, of average height for their age (between 3 and 19 years), in addition to a much larger number of radiographs of children suffering from various pathological conditions, and came to the following conclusions:

a. The dimensions and shape of the sphenoidal sinus are subject to wide individual variations.

b. The chronology of the development of the sphenoidal sinus is graphically represented in a diagram.

c. The sphenoidal sinus is first visible at a fairly constant age, ranging between three and three and one-half years. The limits of the sinus are clearly distinct at about eight years of age. At 16 its posterior limit reaches the plane of the posterior limit of the sella turcica. Normally it does not increase after 16 years of age.

d. In conditions of pituitary insufficiency, congenital or acquired early in life, the development of the sphenoidal sinuses is hindered in variable degree. This underdevelopment is seen in pituitary dwarfism and infantilism, in myxoedema of pituitary origin, and in mongolism. The sphenoid is frequently solid in growth disturbances associated with embryopathies of different type—namely, achondroplasty and other conditions. In cases of underdevelopment associated with disorders not directly connected with the pituitary gland, like rickets and other deficiency conditions, the development of the sphenoidal sinus is the average for the age.

e. The degree of underdevelopment of sphenoidal sinus is, roughly speaking, parallel with the degree of the hypothalamopituitary insufficiency. It constitutes, therefore, one of the indices of this hypofunction, especially in cases of pituitary dwarfism and infantilism. In cases of pituitary myxoedema the underdevelopment of the peripheral skeleton (wrist, knee) is usually more pronounced than that of the sphenoidal sinus.

In mongolism, the underdevelopment of the sphenoidal sinus, very important on account of its frequency of occurrence, is not usually connected, according to their own experience with the degree of the condition.

f. The underdevelopment of the sphenoidal sinus is not exactly parallel with the underdevelopment of the other sinuses. For instance, there may be a marked underdevelopment of the sphenoidal sinus with slight underdevelopment, or even normal development, of the frontal sinus; and more often, an underdevelopment of the sphenoidal sinus may be associated with a marked mastoid pneumatization.

g. The underdevelopment of the sphenoidal sinus is usually associated with other deformities of the skull, such as variations in the size and shape of the sella turcica—extremely hypoplastic, or enlarged due either to congenital hypofunctional tumors (cranio-pharyngiomata) or to conditions marked by internal hydrocephalus.

h. In cases of constitutional or pathological (acromegalic) pituitary hyperfunction the sphenoidal sinus develops intensely, generally simultaneously with the other cranial sinuses.

The final conclusion is that the detailed study of the sphenoidal sinus is absolutely necessary when examining a pathological cranium, especially in cases of growth disturbances.

BACTERIOLOGY.

Disorders of the nose and pharynx constitute a large part of illness in pediatric practice, according to Goldman and associates[6]. It is important to determine whether an infectious or non-infectious process exists in the proper management of the rhinologic disorders. Many nasal problems have as their basis a vasomotor rather than an infectious disturbance, but if an infectious process exists the effectiveness of therapy depends on the knowledge of the infecting agent. One should also know whether the source of infection is the adenoid or the paranasal sinuses.

The authors of this study feel that the best information may be obtained from bacteriologic examinations. Fifty chil-

dren from 4 to 12 years of age were chosen for this investigation. They were all admitted to hospital during the winter months, for diseases other than those of the respiratory tract. Cultures were taken carefully from the nose and nasopharynx using platinum loops, and inoculation took place on blood agar plates.

Twenty of the children had tonsils and adenoids removed previously, but they showed no significant difference in the nasopharyngeal flora from the other patients.

The nasal flora in this study contained chiefly non-pathogenic microorganisms in small numbers. *Staph. Albus B* was present most frequently. Many of the cultures from the normal noses were sterile. The only potential pathogen found with regularity in the nasal secretions was *Staph. Aureus A.* On the other hand potential pathogens may be present in the nasopharynx of normal children. The most frequent one here was *Str. Viridans.* There were no sterile cultures in this group.

For comparison, cultures from 50 patients with nasal infections were examined. These patients were from private practice and out-patient departments. They revealed mainly pathogens on culture and were found in appreciable numbers. The most common organisms were *Pneumococcus* and *Staph. Aureus A;* also, there was a fairly large number of *H. Parainfluenzae.* The series showed that the *Strep. Hemolyticus* is not present in upper respiratory infections or the normal nasopharynx as frequently as is usually thought.

Asked if there were any vaccines now available that are effective in preventing or alleviating the common cold, the British Medical Journal[7] replied as follows:

"No, there are no effective vaccines as far as the common cold infection itself is concerned. Antibacterial vaccines may be of value against secondary infections, but there is no good justification for using them in a widespread manner. No new work has indicated lines on which a successful vaccine is likely to be developed soon. There is no scientific basis for using oral vaccines against colds."

McKee[8] made some interesting observations on the bacteriology and virology of acute infections of the upper respiratory tract:

First, microbes exist as populations when the mixture is not only of different species but of different genera as well. The ratio of one species or genus to another may be important.

Second, certain factors may promote a disparity among the total population. Factors such as diet, pH, and natural microbial antagonism and immunity are mentioned. Antibiotics are frequently the cause of such upsets in the microbial population. For instance *Hemophilus Influenzae* was a relatively uncommon component of the normal oral cavity before the use of penicillin allowed it to be cultured in high incidence.

Third, it is now likely that there are similar interrelationships between bacteria and viruses and between viruses and viruses.

Some bacteria adapt readily to therapy or other would-be adverse situations. Since microbial growth rates are so great the uncommon organism today may thus become the prevalent organism tomorrow.

Many changes in bacteria are dynamic and not at all permanent, so that the ebb and flow of changing circumstances subgest an equilibrium propensity on the part of the microbe. For instance staphylococci may increase from, say, 10 per cent to 60 per cent resistant while on antibiotic. Then if the antibiotic is stopped, this may drop to 40 per cent and then go up to 60 per cent when the drug is started again.

Viruses are a considerably more difficult group to study, but some of their genetic potentialities have been studied and have provided interesting material for speculation. This work is very complex and not possible to summarize except to note that the studies concern the virus reproductive cycle, the antigenesis of viruses, immunity and carrier states.

This is a very thoughtful paper and should be read in its entirety.

DRUGS.

Stark[9] compared hexylcaine with cocaine by animal tests for toxicity and clinical tests for potency, the latter by applying the solutions to the nasal mucosa, observing as well the efficacy of hexylcaine as a topical anesthetic for endoscopic examinations.

Further observations were made regarding the hydrolysis of the drug in human serum.

His conclusions were that hexylcaine appears to be as efficient a local anesthetic as cocaine for endoscopic procedures and appears to have a greater margin of safety.

King[10] reported a case of an allergic infant seven weeks old, in whom the use of vasoconstrictor nose drops was effective in providing temporary airway; but the "rebound" swelling was so great that the nasal airway was almost completely non-existent.

Because infants have a very strong tendency to breathe through the nose this child would become so cyanotic after repeated attempts to get air through its nose that tracheotomy was seriously considered.

The case was successfully handled by treating one nostril only with vasoconstrictor drops. The untreated nostril recovered spontaneously in 12 hours after "withdrawal" of the nose drops, then the vasoconstrictor was discontinued completely.

King[11] presented a series of 176 from 2 to 11 years of age upon whom adenotonsillectomy was performed. To each of 85 cases, 5 mgm. of vitamin K was administered orally three times daily for one week prior to operation. The remaining group of 91 cases received none.

At operation the amount of blood loss was carefully measured. The computed amount of blood lost averaged 52 cc. and this was not significantly affected by the oral pre-operative administration of vitamin K.

Silcox[12] studied the effects of intranasal hydrocortisone alcohol.

The search for another steroid which might be as effective as cortisone and ACTH was made, because these drugs had been shown to have many disadvantages among which were the objectionable complications from the doses required, and the fact that not being readily soluble in water they were difficult to prepare for topical use.

Hydrocortisone has proved to be the most promising of the newer steroids, and the alcohol is the most valuable form.

The studies showed that topical hydrocortisone alcohol can exert a beneficial effect on the allergic nasal membrane even in extremely dilute concentrations. In cases of allergic rhinitis with polyps the effect was often dramatic but sometimes temporary. The solution was of no value in acute rhinitis.

The addition of vasoconstrictor components and antibiotics (Gramacidin, Neomycin and Polymixin) produced a solution which was just as effective as before with the straight allergies, but which was also useful in cases with bacterial infection.

McKendry, Schwarz and Hall,[13] believing that a suitably prepared and formulated ACTH preparation can be effectively absorbed through the nasal mucosa, proved its effectiveness in nine normal individuals. They demonstrated a satisfactory decrease in circulating eosinophils and an increase in urinary 17-ketosteroids following a single dose of 40 I.U. intranasal corticotropin.

Following this demonstration they made therapeutic trials on seventeen consecutive patients with conditions known to respond to ACTH or cortisone. In 14 of the 17 patients definite clinical improvement was observed. Two of the improved patients were children.

Case 1. L.S., a three-year-old boy, had nephrosis for 16 months. He had had excellent results following treatment with intramuscular ACTH on three separate occasions. When severe edema again necessitated treatment, intranasal ACTH, 40 I.U., was administered by insufflation twice daily for eight days. Two days after, profuse diuresis began, and body weight dropped form 48 pounds to 38½ pounds. When discharged from hospital on the fourteenth day the weight was 31 pounds. The clearing of edema which followed intranasal ACTH was at least as complete as that previously obtained from parenteral ACTH.

Case 8. L.N., a boy aged 16 with severe chorea of five days' duration and with a previous history of Sydenham's chorea successfully treated

with *Cortisone*, was given intranasal ACTH, 40 units three times a day for one day, 40 units twice a day for two days and 40 units daily for one week. On the third day the choreiform movements were considerably diminished in frequency and in force, and on the sixth day the only remaining sign was a slight tremor of the tongue. On the seventh day the intranasal ACTH was discontinued with no recurrence of choreiform movements.

The writers state that improvement was satisfactorily maintained as long as the patients continued to take adequate doses of the intranasally administered hormone. No untoward manifestations were observed apart from slight nasal irritation and occasional sneezing in the first few minutes after taking intranasal corticotropin.

ANTIBIOTICS.

The effect of antibiotics on the nasopharyngeal flora of premature infants is discussed by Marie Stoppelman[14] of Boston. The infants in her study came from two nurseries: the Infants Hospital and Boston Lying-in Hospital. The former received admissions to its nursery from other hospitals and homes, and the latter received the infants immediately into its own premature nursery.

Past experience had shown that the routine administration of antibiotics had brought about a substantial reduction in mortality. The number of premature infants studied culturally was 143, and of these, 51 were given antibiotics as follows: eight received Oxytetracycline (Terramycin); 29, combined Penicillin and Streptomycin; and 14, combined Penicillin and Sulfadiazine.

Nasopharyngeal cultures were made on the first, third, fifth and tenth day after birth. In spite of the origins of the two different groups studied, there was no significant difference in the nasopharyngeal flora. Seventy per cent of all cultures obtained on the first day were sterile. The predominating organism on the third day and thereafter was pyogenic staphylococci, with alpha hemolytic and anhemolytic streptococci; gram-negative organisms (largely E. Coli) appeared next in frequency. The occasional presence of other organisms was noted. One significant characteristic found in the nasopharyngeal flora of the treated infants was the reduction in the presence of streptococci; however, the emer-

gence of pure culture of pyogenic staphylococci was noted in those infants treated with antibiotics.

Schwarzer and his associates[15] studied the effect of tetracycline on infants and children from the clinical point of view. They carried out this work in order to determine:

1. The rapidity of absorption from the gastro-intestinal tract.

2. The duration of serum levels after single and multiple oral doses.

3. The diffusibility into the cerebrospinal fluid.

4. The urinary excretion.

5. The tolerance.

6. The therapeutic effect.

7. The establishment of a rational dosage schedule for children.

The drug was administered orally in the form of chocolate suspension in a concentration of 50 mg. per cubic centimeter. Forty-four children ranging from the ages of one month to thirteen years participated in this study. They were all hospitalized patients and were convalescing from various diseases. The final results showed that: Tetracycline is absorbed rapidly into the blood stream after oral administration; therapeutic serum levels of eight hours' duration were produced with a single dose of 6 mg. per kilogram. Further increase of dosage gave a proportional increase of serum concentration. Tetracycline diffused poorly into the cerebrospinal fluid without meningeal inflammation. It is excreted in high concentrations in the urine, and the patients showed a high tolerance to the drug. A remarkable lack of side reactions was observed. The studies indicated that the effective oral dose of Tetracycline is 25 mg. per kilogram per day given at six to eight hour intervals.

CONGENITAL ABNORMALITIES.

Beinfeld[16] gives an interesting account of a case of bilateral choanal atresia in a one-month premature infant. He points out that this condition is an important cause of asphyxia of the newborn. His case was kept alive at birth by means of

a metal airway in the mouth. This airway was kept in for 17 days, during which time attempts were made to correct the choanal atresia. The bony obstruction on the left side was perforated by a No. 2 Lempert type mastoid curette on the second day of life. A catheter was passed through the nasal opening into the pharynx and left in place. On the next day an attempt was made to perforate the right side, but no air could be felt coming through this side after what had seemed a successful attempt with the curette. During this time the baby was fed by a gavage tube passed through the mouth. The nasal tube soon blocked off, and no air could be felt coming through.

On the seventeenth day of life the left posterior naris was again perforated and a larger tube put in. There was now good breathing, and the airway was removed from the mouth. The baby took the bottle for the first time. The tube in the nose was taken out the next day and was left out, as there was a good airway.

Seventeen days later the breathing became bad enough to require the airway in the mouth again. It was obvious that the opening into the nose had closed. The same procedure as previously used was carried out and a tube inserted, establishing once again a good airway. The other side of the nose was freed at this time and tubes inserted into both sides. Eventually, after some minor difficulties, the openings were established on a permanent basis by inserting the tubes at night and leaving them out during the day. At about five months of age the tubes were replaced every third night, and the treatment progressed to the point where the tubes were left out for a month, and replaced once to ensure a permanent opening.

Walker[17] reports successfully correcting congenital bilateral choanal atresia in a two-month-old female child. He used the transpalatine surgical technique outlined by Ruddy, and followed the case for two years.

His conclusions are: the diagnosis of congenital bilateral choanal atresia is made easily and may be suspected in an infant with recurrent cyanosis, feeding difficulty, mouth-breathing and nasal discharge. It is probable that some

infants who have died with the diagnosis of asphyxia neonatorum were in reality victims of complete nasal atresia. A few patients have been known to survive.

Surgical relief of the obstruction is imperative. Postponement of surgery until the patient can tolerate the condition is risky and undesirable. The transpalatal approach is feasible in the young infant, offers surgery under direct vision and does not require special instruments.

One authority had concluded that closure of the palatal wound was unnecessary, because the palatal flap was always sloughed in patients under six months of age. In this case, however, healing occurred by primary intention and, therefore, it seemed advantageous to close the incision. The astute physician caring for the newborn will uncover more cases in the future. The surgeon has an excellent opportunity to improve the survival rate. No previous experience is necessary to handle a case properly.

Everberg[18] reported a case of abnormal course of the cervical part of the internal carotid artery.

It is important to be aware of this anomaly. It is a congenital aberration and, therefore, may be present in young patients who are subjected to operations on the pharynx such as tonsillectomy and adenoidectomy or incision of peritonsillar or retropharyngeal abscesses.

An abnormal course of the artery may be present to a varying degree and may disclose itself as a faint pulsation of the posterior palatine arch or a prominent pulsating vessel deep to the posterior pharyngeal wall.

ALLERGY.

Allergy and infection in rhinitis are discussed by Pepys[19]. He says that they may be associated in three ways:

1. Allergy complicated by infection,

2. Infection predisposing to the onset of the allergic condition;

3. Infection leading to the development of "bacterial allergy".

The complication of allergy by infection is common, due to the disturbed nasal physiology caused by the swollen allergic mucosa. Control of the underlying allergic condition is necessary if there is to be more than temporary improvement from the antibiotics and chemotherapeutic agents.

The influence of infection as a predisposing cause for allergic respiratory disease may be due to a lowering of the clinical threshold, thus making manifest a previously subclinical allergic state. The role of bacterial allergy as a cause of typical allergic rhinitis is not generally accepted. It has not been adequately proven, although it is often suspected on the basis of negative evidence because of the failure to identify the causal allergen.

Allergic rhinitis may appear at a very early age, and the clinical history is most important at all ages. One form is the repeated so-called "colds". These are not accompanied by the sore throat and malaise of the usual infective cold, but are associated with sneezing and rhinorrhea. They may be of short duration and nasal itching is common. Another form of allergic rhinitis is that of the continuously blocked "stuffy nose". This is often aggravated in the morning, and persists for prolonged periods after infective, or what appear to be infective, colds.

The history of allergic disorders in the family, infantile eczema, asthma, gastro-intestinal disorders, all support the diagnosis of allergy. The nasal mucosa is pale, swollen and boggy, though associated infection may alter this for a time. Cytological examination of the nasal discharge can be helpful, since the presence of eosinophiles supports the diagnosis of allergy. Further evidence may be obtained from trials with therapeutic agents such as ephedrine or anti-histaminic drugs by mouth. Relief of the nasal obstruction indicates that this is due to swelling of the nasal mucosa rather than physical bulk of adenoid tissue.

The remainder of this paper is taken up with the investigation and treatment of the allergic state of the child. This includes the usual investigation of various foods and inhalant allergens. Skin testing is of limited use in infancy but has its value later on. Control of environmental inhalant allergens

may be supplemented by injections of the appropriate extract. Anti-histamine drugs by mouth are most useful, but their prolonged use may lead to loss of appetite, general depression and undue sedation. Local vasoconstrictors for nasal application should be used with care, since potent vasoconstrictors may produce a "rebound" vaso-dilatation.

To obtain results there must be an adequate follow-up and a concerted appraisal by pediatrician, ear, nose and throat surgeon and general practitioner.

TONSILS AND ADENOIDS.

Rendell[20] states that under ideal conditions pre-operative sedation for children undergoing tonsillectomy would perhaps not be necessary; however, the importance of avoiding psychic trauma has long been realized. The practice of holding a crying, frightened child and compelling him to undergo something which he instinctively dreads often leaves an ineffaceable memory that quite naturally results in an intense hatred of doctors, nurses and hospitals. On the other hand, ill-chosen premedication may cause depression of the cough reflex and perhaps allow aspiration of blood or vomit.

Barbiturates combined with atropine were in routine use in her hospital. The children fell asleep in bed and knew nothing of their visit to the theater. No difficulty was experienced through depression of the cough reflex. A disadvantage was that nearly all showed marked post-operative restlessness and, to insure their not injuring themselves, needed more nursing attention than was conveniently available. Post-operative "nepenthe" helped the children to settle; however, the giving of central depressants both before and after operation might well have been criticized had accident occurred.

It was decided to observe the effect of methylpentynol on a series of children for tonsillectomy.

Methylpentynol (oblivion) and atropine were given to 152 children for pre-anesthetic medication before tonsillectomy. It successfully diminished pre-operative apprehension in 130 cases and in 17 it was partially successful. Only in five was it a failure.

The drug was readily acceptable to patients, and toxic or unpleasant side-effects were not observed in this series. Protective reflexes were not depressed and post-operative restlessness was but slight. The difficulties of nursing care were consequently reduced. Evidence on amnesia was incomplete, but the impression was gained that it did not occur to any marked extent.

Methylpentynol fell short of the ideal in the following ways: most children were not asleep, amnesia did not occur and, therefore, the patients were conscious of and remembered the induction, which they did not like. Also they saw what went on in the ward. Of the 152 children, 22 were unhappy. Vomiting was not unusual during the induction. After operation there was need of sedation as often as after barbiturate pre-medication, so that methylpentynol, like barbiturates, appeared to be without analgesic properties.

Jongkees[21] in a paper on the psychic effect of hospitalization and surgical interventions on children makes the point that the operation for tonsillectomy is bound to have some psychical influence on the child.

The age of the child and his intellect are undoubtedly of great importance. If the child can be reasoned with quietly and the whole operation and its after effects explained to his satisfaction, the chances are that no bad psychical after effects will occur; but most children can not be so readily psychologically prepared, and it is necessary to make the circumstances as favorable as possible so that the psychical influence will not be disastrous.

If at all possible the child should be convinced of the necessity for the operation, and be given an explanation of the good results that may be expected before being sent to hospital. He should be told that he will stay alone in hospital without his parents. He should be told about the operating room and the anesthetic, and above all he should be warned that his throat will hurt, and that some blood may come out of his nose or mouth.

At hospital the child's questions should be answered as truthfully as possible and he should not be taken by surprise.

On the other hand his protests should be quietly but firmly rejected.

Post-operatively fluids of a reddish color are a big advantage in that if some of the fluid drains back into the glass from the child's mouth, any blood which may have become mixed with it is not noticeable. The coloring of water or milk with blood from the mouth is often a cause of much distress and anxiety.

The stay in hospital should not be prolonged after 24 hours unless really necessary.

This is an interesting paper well worth reading in its entirety.

Out of the morass of speculation concerning the tonsil and adenoid question, Badger[22] states there are a few specific matters to be considered from the viewpoint of the internist.

First, what are the indications for removal of the tonsils and adenoids? He finds the best answer one made by Lyman Richards: "The best results of tonsillectomy are obtained where the symptoms have been clearly referable to the tonsils and associated lymphoid tissue." Conversely, he observes that the further complaints are from the tonsils, the poorer the results are with their removal.

Second, the hazards of tonsillectomy should be no contraindication for surgery when the need is clearly present. Nevertheless the hazards are worth serious thought. To the internist various accidents associated with tonsillectomy are cause for concern when the procedure is considered so trival and when it is used for treatment for reasons that are not always too clear. It is also only fair to admit that the reports from our medical centers of the hazards of tonsillectomy are of the best results that can be obtained in skilled hands, and that the true incidence of deaths and complications in rural areas can never be determined accurately.

As a result of searching the literature he found only three factual and original studies between 1922 and 1952 with information on the hazards of tonsillectomy.

The internist looks with great skepticism upon the benefits of tonsillectomy in the prevention of respiratory infections

except when breathing is interfered with by overgrowth of lymphatic tissue, or when repeated bacterial infections localize in the tonsils or in the tonsillar glands.

In current studies of the common cold and other viral infections "sore threats" are falling more and more into the group of viral infections, and tonsils and adenoids, present or absent, do not appear to play much part in their course or incidence; furthermore the general and less tangible factor of individual resistance and susceptibility to respiratory disease is all too generally disregarded.

The internist believes that poliomyelitis is only one of the neurotropic diseases in which the trauma of tonsillectomy and adenoidectomy should be avoided in epidemics. Encephalitis of any type, influenza of proved type, as well as epidemics of meningococcus meningitis should be signals for the avoidance of any unnecessary surgery on the respiratory or alimentary tracts. The removal of tonsils and adenoids lays bare a large surface of nasopharyngeal tissue where these viruses and organisms are found.

The human body appears to abhor the removal of tonsils and lymphoid tissue of the nasopharynx, at least before the tonsils have stopped growing. This fact is evidenced by the amount of lymphoid tissue that grows back after the larger tonsils and adenoids have been supposedly cleaned out surgically.

The physician should not regard the removal of these organs as a trivial procedure. Rather it is one requiring great skill in anesthesia, in operative technique, and in post-operative care in addition to careful selection based on the needs of the patient rather than the accessibility of the organ itself. It is indeed clear that the internist and the family doctor cannot escape their share of the responsibility with the otolaryngologist in decisions regarding the need and the wisdom of removal of the tonsils and adenoids.

To Eley[23] it has always seemed poor practice of medicine to refer a patient to an otolaryngologist without first contacting him and explaining why his opinion is desired, or why tonsillectomy and/or adenoidectomy are necessary. The previous history of the patient and the possible underlying haz-

ards are really known only to the pediatrician (the family physician to children), and without this information the otolaryngologist may be at a complete loss as to why the patient was referred. The relationship between the two physicians should be on a consulting basis, and only after such an understanding has been established can the question be solved.

The physician is familiar with tonsils that are so large that they force the uvula into the background and, too, there is the adenoid tissue that produces a deep bass sound during sleep. These local conditions, especially those involving the adenoid structures, may, and do, cause serious complications in infants and children.

To him there is no greater or more serious problem than that of possible impairment of hearing. How often the physician is faced with the question of whether the child is mentally retarded or actually handicapped by impairment in hearing, hence appearing retarded. The need of office and hospital services for persons trained in the art of detecting early changes in hearing ability is one of the most important questions faced today.

The theme of the relationship of mastoiditis to chronically infected adenoid tissue is seen less often since the introduction of antibiotics and chemotherapy. Such agents, however, have had little, if any, influence on mouth breathing, the development of the so-called adenoid facies, inability to sleep because of impaired breathing space, and numerous other disturbances readily attributable to such chronic infections. In these conditions the answer is apparent, *i.e.*, the removal of the infected tissue and/or irradiation as necessary.

He believes that under normal circumstances the dangers and hazards of tonsillectomy and adenoidectomy have been reduced to a minimum when directed and performed by competent physicians.

His conclusion is that most pediatricians view the tonsil and adenoid question with respect, but they also view the general health of their patients with a greater respect, and in

selected instances and under proper circumstances advocate the removal of these lymphoid threats.

Hoople[24] states that now tonsillectomy and adenoidectomy is being performed to conserve hearing much more than in previous years.

In the Northeast, at least, the winter season is the dread of most otologists. This is the time of the year when the office is often filled with children who have inflammation or infection of their ears. When it is the former, it is almost always accompanied by the collection of some sterile fluid in the ear, that is, otitis media with effusion.

This condition frequently is more difficult to treat and eradicate than infection, and neglect can result in partial, but permanent, loss of hearing. Too often children with inflammation and fluid in their ears do not come to the otologist. They remain in the office of the pediatrician, with the accompanying hearing loss undiagnosed or excused with the assumption that the child's lack of response to queries or commands is due to inattention and not to impairment of hearing; furthermore, the end-result of antibiotic treatment for an ear infection may be a sterilization of the infectious process with a residual otitis media with effusion. This is particularly true, in his experience, with penicillin therapy.

The end-result in such an instance is the same as though there were no infection in the first place, but only inflammation. With the subsidence of pain, temperature and discharge (if such were present), the pediatrician is apt to consider the patient cured, although this is far from the truth. Fortunately, some pediatricians are sharply aware of this problem, but there are too many who do not concern themselves with moderate or slight hearing losses. The otologist can do nothing but sound this note of warning.

If, then, the otologist is confronted with a patient who has some of these ear problems, it is his duty to advise a very thorough removal of the infected lymphoid tissue present. If infected tonsils are present, their removal should be included, but the careful removal of the nasopharyngeal disease is the

more important. It does not matter what age the patient may be.

He has removed adenoids from a child only three months old, and has seen infected adenoids in persons who are in their sixties. The time factor is important. Removal should be undertaken as soon as the local condition in the nasopharynx warrants surgery. Waiting for summer is often unwarranted, for the public has become so poliomyelitis conscious that one cannot operate during the summer and early fall months.

A cogent reason why operation should be instituted as soon as possible is that another episode may be just around the corner, and this episode may be the one that will really cripple the hearing in one or both ears. Each attack is potentially a serious one. Every attack probably lays the groundwork for some loss of hearing, but one never knows which attack may leave a striking aftermath.

He advocates adenoidectomy under vision and for this prefers a Lothrop retractor and a triangular forceps with a basket attachment.

Secondary adenoidectomy he considers more difficult than the original adenoidectomy. Infected lymphoid tissue bound down by scar tissue is often present. No curette and no adenotome will remove this. It must be cut away with a proper biting forceps, and this must be continued until all lymphoid tissue is removed. A secondary adenoidectomy often takes longer to perform satisfactorily than the primary one.

One comforting thought to the operator who follows this technique is that he will have less post-operative bleeding from the nasopharynx. Most nasopharyngeal bleeding comes from incompletely removed lymphoid tissue.

He failed to obtain satisfactory results from the use of the radium applicator. Since he has paid more attention to the complete adenoidectomy, his surgical results, the decrease in the incidence of ear infections and the improvement in hearing in these patients have been gratifying.

Boies[25] states that surgery of the tonsil, discussed for nearly 3,000 years, is still controversial, probably first because

the indications for its surgical treatment are often based on superficial observation; and second, because there is a morbidity and even mortality associated with it, since it is attempted by many who have not acquired skill through training and experience in working in this area.

Tonsillectomy and adenoidectomy is justified in cases of repeated attacks of tonsillitis or adenoid infection, hypertrophy to the extent of obstruction, and when there is evidence of persistent chronic tonsillar or adenoid infection. A clean tonsillar fossa with the minimum of trauma to the pillars and muscular bed and thorough hemostasis are the marks of a skillful, carefully performed tonsillectomy.

A thoroughly performed adenoidectomy requires good exposure of the nasopharynx, time, hemostasis and visualization of the end-result. The hazard of hemorrhage is minimal if care and thoroughness are used. The anesthetic risks of periods of anoxia, uncontrolled aspiration of secretions into the lung, and other anesthetic hazards are practically eliminated with the best modern intratracheal method.

The hazard of increased susceptibility to bulbar poliomyelitis and of the greater possibility of death, if the disease is contracted, "call for sober judgment with regard to the desirability of tonsil removal except for excellent clinical indications. The non-removal of tonsils may be as hazardous to a person or more so than their removal, for the chance of contracting poliomyelitis in a clinically recognizable form is not great".

In the Section of General Practice of the Royal Society of Medicine[26] a discussion was held on tonsils and adenoids.

One speaker stated that the problem of enlarged tonsils and adenoids was mainly one of childhood. Lymphoid tissue tends to regress after the age of 12 to 15 years. After that age there is no problem of treatment. "When in doubt, take them out." They have served their purpose and can be dispensed with if there is reasonable evidence of their doing any harm.

He believed that the indications for the removal of ade-

noids were straightforward and the decision easy to make. The indications were:

1. Gross nasal obstruction producing constant mouth breathing and snoring when asleep, even when there was no coryza present.

2. Recurrent attacks of otitis media with or without otorrhea.

The problem of how best to deal with diseased tonsils was more difficult. Probably all practitioners had their own ideas of what a chronically infected tonsil looked like, but it was extremely difficult to prove the infection and its chronicity. The following he considered definite indications for the removal of tonsils in children:

1. Frequent attacks of tonsillitis with persistently apparent infection in the tonsils between attacks and persistently enlarged and tender lymph nodes.

2. After two and possibly after one quinsy.

3. When the tonsils are so large as to cause obstruction of respiration.

4. Diphtheria carriers.

Another speaker added three diagnostic points to the above indications for adenoidectomy. The first was that chronic sinus infection was commonly linked with enlarged adenoids. The second was that a child who was sick in the early mornings before breakfast, with a slimy vomit, had probably been swallowing mucopus all night and needed attention to the adenoids; but neither of these associations was invariable and absolute. The third was a diagnostic warning. The child brought with a history of frequent absence from school because of colds, of much early morning sneezing, and of vigorous nose-blowing with very little result, was suffering from allergic rhinitis, for which operation on tonsils and adenoids was useless.

He advised caution regarding tonsillectomy. He stated that a child who failed to thrive, or lost its appetite, might benefit from tonsillectomy. The operation was valuable in the treatment of tuberculous glands of the neck, because the tonsil

was usually the portal of entry of the tuberculosis. Tonsillectomy should be done whenever tonsillar disease impaired the child's health, no matter what his age.

A third speaker pointed out that it was physically impossible to remove adenoids completely. When the tonsils were completely removed, particularly in the first five years of life, lymphoid tissue around the pharynx tended to hypertrophy as a substitute for regeneration. This tendency was a further reason for conservation rather than operation.

Johnston and Watkins[27] claim that during the past 30 years there has been an increased conservatism in the decision to remove tonsils and adenoids, but they feel that the pendulum has swung too far to the conservative side. The authors' appraisal of results following the removal of tonsils and adenoids in 598 children was arrived at by:

1. Replies from parents as to their opinion on the effect of the removal on specific complaints.

2. By rechecking several groups of patients with conditions thought to be influenced by chronic infection.

3. By a small series of metabolic studies before and after the procedure. Of the 598 children, 112 were between the ages of one and four years, 331 between the ages of five and seven and 155 between the ages eight and fourteen.

Improvement was noted in 71 per cent of ear infections; 89 per cent of the cases with abdominal pain referable to chronic infection were improved. Out of 87 patients of all age groups with bronchitis, 74 showed improvement. Of those patients with sinus infection only 13 out of 28 were benefited by the operation.

The results of tonsillectomy on patients with asthma were unsatisfactory, as only three out of 22 asthmatics showed any improvement. Children with anorexia showed an improvement in 84 per cent. Impaired hearing was benefited in seven out of nine patients, and eight of ten cases with infectious arthritis were improved.

The metabolic studies showed nitrogen retention was better in nine children. One child, followed for 18 days before and

27 days after operation, showed a three-fold increase in the storage of both nitrogen and calcium.

As a result of these findings the authors conclude that tonsillectomy and adenoidectomy remains a highly beneficial procedure in the properly selected cases.

Works[28] discussed the complications of tonsillectomy, and reported on the experience of the Ross-Loos Medical group with more than 28,0000 tonsillectomies.

He feels that adenotonsillectomy should be considered a major surgical procedure and stressed the need for positive indications for surgery.

The locations of tonsils makes surgery on them risky. Among the complications that can occur are aspiration, spread of infection into the neck, and hemorrhage. The tonsils are in a vascular area and are also in a muscular mobile area which tends to dislodge clots and ligatures, and to release thrombi; furthermore, the fact that blood can be swallowed may conceal hemorrhage.

The number of complications in this series was small, likely due to careful pre-operative work-up, insistence on highly trained surgeons, and anesthetists plus recovery rooms with close supervision and facilities to handle any emergency that might arise.

The paper includes useful information on the management of complications if and when they do arise.

Meltzer[29] concludes an admirable paper on the technique of removal of adenoid tissue of the nasopharynx in children by saying that the technique described has given him great satisfaction when used for children in whom tubal closure with hearing loss was a recurring problem. Regardless of the specific purpose for which this technique can be applied, it is high time for otolaryngologists to show the correct way to perform an adenoidectomy, thereby saving countless numbers of children from the miseries of recurrence.

Adenoidectomy is generally considered to be a minor surgical procedure of little consequence. The technique used may be deplorable, yet the results may be amazingly good. For this

paradox, nature is to be thanked. The natural process of recession accounts for the excellent results we see.

The record is not so good in patients presenting a complicated problem, such as sinusitis or otitis, or in patients in whom re-growth and hypertrophy develop after removal of the original obstructing tissue. Remnants of tissue left behind in the fossa of Rosenmuller, or on the Eustachian tube—such tags of tissue can and do undergo edema from infection causing recurrent tubal closure, with subsequent secretory or suppurative otitis media and ultimate hearing loss.

A great step forward toward visualizing the surgical area was made when Yankauer introduced the tubular speculum, and it remained for Lothrop to provide a retractor that made it possible to visualize the nasopharynx and the structures in it.

The anatomic details of the postnasal space and the variations cannot be appreciated and taken fully into account unless the palate is elevated and the area actually inspected.

Only experience will perfect one in the art of elevating the palate properly. To expose the torus, the retractor is placed laterally on the palate just above the tonsillar fossa and the wrist flexed. Slipping the lip of the retractor behind the torus and again flexing the wrist will beautifully expose the fossa of Rosenmuller.

To visualize the postnasal space in some cases requires extreme pressure on the palate. The ease with which the entire postnasal space can usually be exposed to view makes it inexcusable for the otolaryngologist to carry out adenoidectomy in any but the careful and thorough manner possible only under direct visual control. Once the main mass of adenoid tissue is inspected it can be removed by several insertions of a sharp-bladed LaForce adenotome of suitable size.

Next one inspects the Eustachian tube, the lateral bands (salpingopharyngeal folds) and the fossa of Rosenmuller. The surgeon who has never performed any but "blind" adenoidectomies has a surprise in store for him. A direct view of the anatomy of this region will make it clear to

him at once why neither curette nor LaForce adenotome alone can remove lymphoid tissue from this area.

He will find that the fossa of Rosenmuller varies widely in depth and in width. Sometimes the fossa is so deep that one cannot see the cutting edges of the instrument, no matter what retractor one uses. One learns to tell by the feel of the instrument when the recess has been cleaned of lymphoid tissue. In this area he uses a No. 1 ring punch, with a gentle curved sweeping motion of the instrument, opening and closing it repeatedly along the superior margin of the posterior nares as it blends with the upper portion of the fossa of Rosenmuller.

In the relatively few cases in which the surgeon cannot see the head of his cutting instrument he may have to palpate this particular area for small tags. He utterly condemns as non-surgical the maceration of adenoid tabs by friction with a gauze-wrapped finger. To remove the lateral bands lying along the posterior pillar of the tonsil extending upward to the tube, he gently rolls the pillar laterally. The hypertrophied band of lymphoid tissue is then trimmed to the desired depth. He disapproves of sharp dissection or stripping of this tissue. Lymphoid nodules on the posterior wall are removed with the small oval punch, cutting with the pharyngeal surface. Deep bites may produce unnecessary scarring.

Even under direct visual control islands of lymphoid tissue will remain in the mucosa that may subsequently hypertrophy.

The removal of recurrent adenoid growth makes greater demands on the surgeon's skill than a first operation. Because of bleeding, second removal is a time-consuming procedure that calls for great care and patience. He places sponges in the nasopharynx to control bleeding and also uses curved hemostats to clamp the vessels.

As a final precaution he paints the entire surface of the post-nasal space with Garland's solution (tincture guiac 25 cc; tincture iodine 25 cc; and tincture benzoin compound 200 cc).

Fisher[30] has discovered that post-nasal adhesions are a common occurrence and, believing that this fact is not generally known, he desires to attract recognition of its importance as a factor in diseases of the ear, nose and throat.

He states that it cannot be doubted that the conditions of the lateral pharyngeal recess are ideal for the implantation, harborage and establishment of pathogenic infection, and that the clinical findings so commonly met with, i. e. presence of adhesions binding the walls of the lateral recess are surely proof of previous or persisting inflammation.

The author breaks these adhesions with his right index finger with the patient lying recumbent. With high strung and hysterical individuals and children, a brief and light anesthesia is required. The performance is followed morning and night by a gravitation bland saline nasal irrigation.

He devotes a paragraph to the clinical manifestations and gives brief descriptions of four patients who were relieved by finger manipulation.

Comment: The condition of the nasopharynx is best discovered by nasopharyngoscope. Although rupture of the adhesions has frequently benefited the patients, the writers have often seen adhesions which were not causing the patient any disability. The condition has long been recognized in America and has on occasion led to sporadic waves of spurious treatment.

Reeves and Brill[31] reviewed the anatomy and histology of Rosenmuller's fossa, the tubal orifice and the folds around the tubal orifice in 622 visually controlled adenoidectomies. There are three folds around the tubal ostium which if enlarged may interfere with the free movement of air to the middle ear.

The salpingopalatine fold accentuates the anterior lip of the tube and is reinforced by a ligament. This plica varies in thickness according to the number of glands and lymph follicles in the mucosa covering it.

The salpingonasal fold arises from the ostium and is directed upwards towards the roof of the choana. It may be separate from or be a continuous plica with the salpingopalatine fold.

The plica salpingopharyngea arises from the posterior lip of the orifice and continues downward behind the posterior pillar.

Histologically these folds are similar to the pharyngeal tonsil at its inferior border.

These plica are best observed with a laryngeal mirror after the soft palate has been retracted with a catheter.

If they are wider than 5 mm. the writers remove them with Meltzer punches.

In the Section of Laryngology of the Royal Society of Medicine[32] a very long discussion was held on the management of hemorrhage following tonsillectomy. Almost every aspect of this subject was covered in a well-planned survey. Reference to any question is easily found. The reader is referred to the original.

Comment: Some text books current on this continent give advice regarding this complication which appeals to your authors as both shorter and sounder.

The Lancet[33] commented on certain parts of this discussion.

Ashcroft had heard within the past few weeks of no fewer than six deaths from this cause. Hemorrhage after tonsillectomy was terrifying to the patient. It might be equally alarming to a doctor inexperienced in dealing with it. More skill was needed to control hemorrhage than to do the tonsillectomy. Successful treatment depended on an understanding of the causes.

He mentioned many undesirable preoperative factors, the significance of which should be discussed fully with a competent physician before tonsillectomy. Skill and care in doing tonsillectomy could certainly reduce the incidence of postoperative hemorrhage. The tonsillar fossae must be dry before the patient left the theatre. On completion of the operation, all blood clot should be removed from the nose and nasopharynx as well as from the tonsillar fossae. The patient should be placed on his side and the tongue prevented from falling backwards.

One might expect slight oozing for the first hour after operation; but if, after this, there was still bleeding or if freshly swallowed blood were vomited, the case should be treated as one of reactionary hemorrhage. If certain minor methods such as pressure, and removal of blood clot, failed to stop the bleeding, the patient should be returned to the

theatre without delay. Bleeding points should be identified and ligatured.

True secondary hemorrhage, associated with damage to the muscular tissues of the tonsil bed, occurring any time between the third and fourteenth day, and accompanied often by a raised temperature due to the local inflammation, was fortunately less common. Since the infected tissues were friable, simple ligature was often difficult.

Mr. S. Mawson felt that, apart from stopping the bleeding, the replacement of blood volume was the vital factor in saving life in the reactionary period. The volume of blood that could be safely lost without replacement depended on the age of the patient; it should not exceed one-tenth of his total blood volume. Replacement was essential if the blood loss reached three-tenths of the total volume.

Comment: The chief speaker stated that suturing of the faucial pillars over a gauze swab was sometimes necessary in cases of true secondary hemorrhage where the infected tissues were friable. This method for obvious reasons is condemned in good otolaryngological teaching on this continent.

Cummings[34] discussed the mortalities and morbidities following 20,000 tonsil and adenoidectomies performed in a 30-year period by general practitioners and otolaryngologists.

There were nine deaths, four from hemorrhage, three from cardiac arrest and two anesthetic deaths; also, there were seven lung abscesses, three foreign bodies, plus a number of minor morbidities.

From a study of this series he made the following observations and recommendations: Internes on rotating services should be taught to perform tonsil and adenoidectomies, and that residents in otolaryngology should spend at least three months in the department of anesthesia. Drop ether is the safest anesthetic for this operation. Solutions of cocaine in the operating room should be colored and should be in charge of the department of anesthesia.

He also felt that all patients who bleed postoperatively should have a hemoglobin determination and a transfusion if their hemoglobin is below 60 per cent. The head-low position should be used for operation, and the patient should be kept

in the operating room until he has reacted. Then he should be accompanied to the ward or recovery room by the anesthetist.

Close postoperative watch should be kept by the nursing staff. He felt that intratracheal anesthesia should be limited to adults because of the danger of postoperative laryngeal edema in children. No preoperative sedation with depressant drugs should be used in children. When cardiac arrest occurs the chest should be opened for cardiac massage. Allergy *per se* is not a contraindication to tonsil and adenoidectomy. Finally, that the possible relationship of poliomyelitis to tonsil and adenoidectomy be further studied.

Emerson[35] describes a technique by which after removing the tonsil he sutures the mucosa of the anterior pillar to the bed of the tonsil fossa and the posterior pillar. By this he controls hemorrhage, eliminates dead space and covers the denuded surface to allow healing by primary intention.

He has performed the operation on 215 patients whose ages ranged from 18 months to 60 years. The operating time is about 20 minutes for children and 30 minutes for adult patients.

The healing time in children is five to seven days. Adult patients have returned to work on the seventh to tenth postoperative day without difficulty. There have been no serious complications; hemorrhage has not occurred; infection has not been a problem, although 300,000 units of procaine penicillin is used routinely on the day of operation and the following day, except in patients known to be sensitive to the antibiotics.

IRRADIATION.

Hardy and Bordley[36] reported on the five-year joint research project started in 1948 by the Johns Hopkins School of Hygiene and Public Health, the Department of Otolaryngology of the Johns Hopkins School of Medicine, the Department of Health and the Department of Education of the City of Baltimore.

They report the following observations from a controlled

study on the effect of nasopharyngeal irradiation in a group of school age children:

1. There is unmistakable evidence that nasopharyngeal irradiation reduces lymphoid tissue to a remarkable degree.

2. Both the treated group and the control group showed improved hearing. The improvement was always more marked for the treated children whose hearing loss was a conductive type affecting all tones. The improved hearing in the untreated children can be accounted for only by factors such as puberty, learning and improved nasopharyngeal hygiene.

There was no evidence of trauma relatable to irradiation of the nasopharynx.

3. The physiologic improvement in hearing takes place at puberty. This may be associated with the change in the condition of the lymphoid tissue. No other finding is so consistently relatable.

4. The modern diagnostic methods are efficient in predicting hearing loss in school population.

5. The success of this study was clear evidence of the feasibility and effectiveness of inter-agency cooperation in a school health program.

Meltzer[37] is studying lymphoid tissue in the nasopharynx. This tissue may be described as a mass of free cells, mainly lymphocytic, of various sizes, plus a supporting framework of reticulum cells and fibrous, elastic and sometimes muscular elements.

The functions of lymphoid tissue has not yet been established. Some believe that along with the tonsils it guards the gateway to the alimentary canal. The defense theory further suggests: *1.* That lymphoid tissue filters undesirable material from the circulation; *2.* That antibody formation takes place in the lymphocyte.

To conciliate the barrier concept with the clinical experience that tonsils often become foci of infection and have to be removed, it has been suggested that these tissues, evolutionarily speaking, are a recent acquisition which is unequal to the assaults occasioned by exposure to crowded urban life.

Lymphoid tissue appears to be affected by allergy and in these patients there is a great tendency to recurrence even after the most thorough surgery.

The endocrine factor promises to be an important avenue of research. Adrenocortical secretion causes lymphoid tissue involution. It may be that further experience with ACTH and Cortisone may indicate their use in nasopharyngeal lymphoid hyperplasia.

At present the treatment varies from patient to patient, but may require surgical removal, irradiation or allergic management, or combinations of these three measures.

TONSILS AND POLIOMYELITIS.

Weinstein, Vogel and Weinstein[38] studied the records of 800 cases of poliomyelitis at a Boston Hospital to see whether tonsillectomized patients showed an increased susceptibility to bulbar poliomyelitis. This work had been done before, but the authors were anxious to repeat the investigation to see whether it applied to patients observed by them.

Of the 800 cases studied, 500 individuals had been subjected to tonsilloadenoidectomy at one time or another, and 300 had not been operated upon. The types of poliomyelitis were listed as *a.* nonparalytic; *b.* spinal paralytic; *c.* bulbar; and *d.* bulbospinal. The case histories showed that the absence of the tonsils, even though they may have been removed many years before, was associated with a greater incidence of bulbar poliomyelitis. In fact, all forms of poliomyelitis appeared to be more common in the tonsillectomized patients.

The age group ran from six days to 56 years and there were 55.5 per cent males and 44.5 per cent females. Age and sex were not important in conditioning the susceptibility of tonsillectomized patients to bulbar poliomyelitis. They were unable to separate those who had had recent tonsillectomy from the other cases, so they were evaluated in terms similar to the general tonsillectomized patient.

The authors showed that bulbar poliomyelitis was three to four times, and the bulbospinal type three to five times more common in tonsillectomized cases than in those who had not

been operated upon, regardless of age. The observations made in this study confirmed those of other investigators.

The *Journal American Medical Association* commenting[39] editorially on the relation of absence of tonsils to poliomyelitis, states that data have been provided over a number of years to suggest that mere absence of tonsils and adenoids, regardless of the time of their removal, leads to increased susceptibility to poliomyelitis. Three reports are summarized, the latest one being from Haynes Memorial Hospital, Boston, in which 800 patients with poliomyelitis were studied.

This study suggested that the absence of tonsils and adenoids, regardless of the time of their removal in relation to the time of onset of poliomyelitis, increased the risk that the bulbar form of the disease would develop. Of 85 patients in whom bulbar poliomyelitis developed, 85.9 per cent had previously been subjected to tonsillectomy; of those in whom tonsils were still present, bulbar poliomyelitis developed in only 14.1 per cent.

The incidence of both non-paralytic disease and spinal paralytic disease was higher in patients without tonsils than in those with tonsils. Also, the data presented by the authors appeared to suggest that the date of tonsillectomy when related to the onset of poliomyelitis may be of less significance than the mere fact that tonsils were absent.

Settlement of the latter aspect of the tonsil-poliomyelitis problem is of considerable importance to the entire medical world and merits further study, in view of the growing belief that recent tonsillectomy may lead to increased susceptibility to the bulbar and bulbospinal forms of poliomyelitis.

The relation between tonsillectomy and poliomyelitis, and between the presence or absence of tonsils and poliomyelitis, has been the subject of discussion for some years. From time to time equally forceful, but in some respects conflicting, views have had ardent supporters, and even in the *Journal American Medical Association* different views on the subject have appeared within weeks of each other.

A careful analysis of these and other published reports will show, however, certain basic agreements, even though

there may not be a meeting of the minds on other points. One of these beliefs about which there is less disagreement concerns the time interval between tonsillectomy and adenoidectomy and onset of the disease. Most otolaryngologists seem to agree that if poliomyelitis strikes within a matter of days after such an operation there is a greater risk that the patient will have the bulbar type and more severe illness; and most of them also seem to have sufficient apprehension to recommend these operations only on a selective basis during epidemics of poliomyelitis.

It is obvious that more study is needed on this important problem. Some facts are now apparent, others still in question. While a determination of the true relationship between some operations (or the effect on the loss of tissue by these operations) remains for further exploration before all of the questions are answered, one ancillary observation emerges. Once again is there ample demonstration of the need for asking, "Is this operation really necessary?" before the tonsils or adenoids are removed.

Juers[40] criticized the preceding editorial as follows: "In the April 3, 1954, issue of the *Journal* there was an editorial entitled 'Relation of absence of Tonsils to Bulbar Poliomyelitis'. It was based largely upon a statistical study by Weinstein, Vogel and Weinstein *(Jour. Pediat.*, 44:14, Jan., 1954). These authors inferred that the absence of tonsils and adenoids increased susceptibility to the development of bulbar and bulbospinal poliomyelitis. The validity of the statistical study is not questioned, but in this, as in many other recent similar reports, the conclusion reached is on somewhat fallacious grounds.

"The fallacy is that the incidence and severity of a given disease are being compared in two biologically and immunologically dissimilar groups of persons. In general this group not having had tonsillectomy has had better immunity to disease, especially in the upper respiratory area, and hence has not been subjected to tonsillectomy. The second group, on the other hand, has been subjected to tonsillectomy because of poor immunity to disease, particularly in the

cephalic area. This poor immuno-logic response would undoubtedly also apply to invasion by the poliomyelitis virus.

"It is my clinical impression that there is usually a greater degree of autonomic instability in the cephalic area in these persons before tonsillectomy, and that as a result the area may have become "conditioned" to respond in an abnormal manner to many forms of stress, probably including that of poliomyelitis virus invasion.

"The unanswered question still is whether the higher incidence of bulbar poliomyelitis in patients who have had tonsillectomy is the result of the absence of the tonsils and adenoids *per se*, or the result of pre-existing, greater local susceptibility to the poliomyelitis virus as compared to that of patients who have not had tonsillectomy."

Molenaar[51] comments about the editorial *(J.A.M.A.*: 1180, April, 1954), supporting the thesis that tonsillectomy increases the incidence of bulbar poliomyelitis, which compares persons who have had presumably abnormal tonsils removed with persons who have had presumably normal tonsils left in: "To avoid the obvious, that sick persons get sick more often than well persons, one should compare those who have had abnormal tonsils removed with those who have had abnormal tonsils left in".

Player[42] states that the data collected in the many surveys which have condemned tonsillectomy in the development of poliomyelitis do not lead to that conclusion. The surveys have never really utilized a control group.

He maintains that the surveys so far conducted have merely contrasted the incidence of bulbar poliomyelitis in patients with relatively more than average lymphoid disease (in whom the surgery is literally only an index of the severity) with the incidence in patients who have not had lymphoid disease sufficient to warrant operation. In other words, from the data collected so far there is even as much reason to conclude that if these severely involved patients had not had tonsillectomies done they might be even more susceptible to bulbar poliomyelitis; or, stated otherwise, surgery may well be protecting some patients from susceptibility.

Anderson and Rondeau[43] state that foremost among the many unknowns in the pathogenesis of poliomyelitis are the factors that determine the type of the response to the infection. There is ample evidence that infection with the poliomyelitis viruses is widespread, ultimately involving almost all persons except those in isolated areas. In most of these persons the infection is mild, producing no paralysis and usually occurring without recognizable symptons. Yet a small number of persons respond badly to the virus invasion, with resultant paralysis.

In 1941 Top and Vaughan, reviewing 521 cases of poliomyelitis, had showed that 27 per cent of 215 persons who had had tonsillectomy at some time in their life had a bulbar involvement in contrast with only 4.2 per cent of 306 persons whose tonsils were *in situ* at the time of the attack. Anderson and Rondeau, therefore, studied 2708 epidemiological histories gathered during the 1946 outbreak of poliomyelitis in Minnesota in an attempt to confirm the observations mentioned and to explore the causative factors.

This paper is very long, provided with ten tables (errors in two of which were subsequently corrected), and bears the impression of exactitude, fairness and careful analysis. It terminates with valuable comment to which the reader is recommended.

Their summary and conclusions are as follows:

"It has been found that persons with the bulbar type of poliomyelitis give a history of removal of the tonsils more frequently than do persons with other forms of poliomyelitis, and that, if clinically recognizable poliomyelitis develops in a person who has had his tonsils removed, the likelihood of bulbar involvement is four times as great as one whose tonsils are *in situ*.

"This higher proportion of cases of bulbar involvement in patients who have had tonsillectomy occurs at all ages regardless of the time that has elapsed since operation. This higher proportion in older persons is due primarily to absence of the tonsils rather than to age *per se*.

"The higher proportion of bulbar involvement in recent

years may be due to the greater frequency of tonsillectomy and the shift in age distribution of poliomyelitis to the ages at which tonsils may have been removed. The lack of cases of bulbar involvement in certain areas may be due to the concentration of poliomyelitis in ages before tonsil removal. The hypothesis that bulbar involvement is due to escape of the virus from the blood stream through the capillaries of the medullary area is not compatible with the greater incidence of such cases in persons who have had tonsillectomy.

"It is possible, however, that the excess of bulbar involvement in persons who have had tonsillectomy is due to direct invasion along fibers of the ninth and tenth cranial nerves. No evidence was found as to the effect of tonsillectomy in altering susceptibility to clinically recognizable poliomyelitis. The data presented deal only with likelihood of bulbar involvement if clinically recognizable poliomyelitis develops."

Robinson[44] believes that the statistics given in "Absence of the Tonsils as a Factor in the Development of Bulbar Poliomyelitis," by Anderson and Rondeau, may be misleading or the interpretation of them may be. Could it not be that the very conditions that caused the tonsils to be removed favor the bulbar type of poliomyelitis? If we accept the conclusion that removing tonsils favors the development of the bulbar type, many who need their tonsils removed may be deprived of the benefit.

A correspondent asked the *Journal American Medical Association*[45] two questions: *1.* Is there any objection to giving a patient the Salk polio vaccine one week after tonsillectomy? *2.* Is tonsillectomy contraindicated within one to two weeks after the patient receives the Salk vaccine? He received the following reply:

"There is substantial evidence of an increased risk of poliomyelitis, especially bulbar, during the immediate postoperative period following tonsillectomy. This short-term risk has been documented only for patients operated upon during epidemics of the disease. There is also evidence, however, of a relatively high incidence of bulbar poliomyelitis in persons subjected to tonsillectomy at longer intervals before

onset of the disease. No reason is evident why administration of the Salk vaccine following tonsillectomy should be expected to increase the risk of poliomyelitis.

"The vaccine is treated with formaldehyde to inactivate the poliomyelitis virus, which is its essential constituent. The tests employed to assure completeness of inactivation appear adequate to exclude any residual infectivity of the vaccine. It is true that during epidemics of poliomyelitis some correlation has been found between the site of paralysis and the site of recent injections of various non-viral agents. Conceivably an inactivated poliomyelitis vaccine, administered to someone already infected, might be capable of such a non-specific effect. There is no evidence so far, however, that this is the case."

MacGregor[46] reports that a boy, age three, fell over while carrying a stick in his mouth, and the end of the stick forcibly enucleated his right tonsil. Seventeen days later he became ill with what was proven to be poliomyelitis. He made a stormy recovery.

Hunter[47] describes an almost exactly similar case in a boy, age four. Poliomyelitis developed seventeen days after he lacerated his right tonsil while falling with a stick in his mouth. Recovery was slow due to persistence of facial weakness.

MOUTH AND THROAT INFECTION.

McMath and Pereira[48] make a case report on a 10-year-old girl who died from the rupture of an undiagnosed retropharyngeal abscess.

After an illness of a week this child was admitted to hospital exhibiting mild stridor, nasal speech, marked cervical adenitis and periadenitis in the anterior and posterior triangles. The post-pharyngeal wall was inflamed and edematous with no evidence of abscess formation. She swallowed fluids without difficulty.

Eight hours after admission she suddenly became cyanosed and rigid, and stopped breathing. Tracheotomy was at once performed and a tube inserted through which a large amount

of thin brownish fluid exuded from the trachea. She died immediately.

Post-mortem examination showed an extensive abscess occupying the posterior pharyngeal region from the uvula and tonsillar region down to the arch of the aorta. It had recently ruptured into the pharynx and flooded the trachea and bronchi.

TUMORS.

Gundrum, Stambuck and Gaines[49] had a newborn that presented the problem of obstruction to both nasal breathing and swallowing.

They found that by raising the soft palate a grayish movable mass was exposed which lay against the posterior pharynx. No fluid was obtained by aspiration.

The mass was pedunculated and was removed by tying the pedicle and cutting it with scissors.

Histologically the mass was covered by epidermis and contained well-formed skin appendages such as hair follicles, sweat and sebaceous glands, as well as fatty tissue in the center. It was classified as a choristoma. These are a displacement of tissue in the course of development.

A case of asphyxia neonatorum due to a nasopharyngeal teratoma is reported by Kesson[50]. This baby was a normal delivery, but three hours after birth he suddenly became cyanosed and dyspneic. Examination showed a yellow mass in the oropharynx, and when the baby gagged, a sausage-shaped tumor was brought forward into the buccal cavity, pulling the soft palate anteriorly. A rubber catheter was passed into the pharynx beyond the tumor to provide an adequate airway. The pedicle of the mass was then snared and the tumor removed whole without any anesthetic. The relief from respiratory embarrassment was immediate.

The specimen measured 2x1x1 cm. It was apparently covered by skin and there was a narrow pedicle. Examination of the cut surface showed the center of the nodule to be occupied largely by adipose tissue. Microscopically there was a small nodule of cartilage at the center and surrounding this

a broad layer of adipose tissue. The exterior of the nodule was covered by skin which showed well-marked keratinization and numerous pilo-sebaceous follicles. The nodule was clearly a congenital malformation.

Schindler, Hurwitz and Greenhood[51] reported the case history of a newborn with a nasophyngeal tumor at birth which caused the baby to have an unusual amount of mucus in the nose and throat, and made the infant choke and become cyanotic at first feeding.

Examination disclosed a small white polypoid mass protruding about 3 mm. from underneath the left posterior portion of the soft palate. It was found to be attached by a pedicle to the region of the left fossa of Rosenmuller. Whenever the child attempted to swallow, the mass would be drawn down and would obstruct the airway.

The mass was removed with snare after first ligaturing the pedicle at its base. No anesthesia was necessary. Thereafter the child did well and at the age of four and one-half months there was no evidence of the tumor.

Histologically the mass was covered by squamous epithelium, and numerous skin appendages were present including sebaceous and sweat glands, hair follicles, and fat and cartilage as well. Such a teratoid tumor in the newborn is extremely rare.

Cassidy and Wahl[52] presented two cases in which central nervous tissue occurred in nasal tumors of infants.

These are difficult to differentiate from encephaloceles and meningoceles, which have central connections. The test described by Furstenberg may be helpful. In this test, pressure on the homolateral jugular vein will cause pulsating expansion in cases with intracranial communication but not in those without.

These tumors are probably the result of congenital embryonal rest of displaced neural tissue.

Nasopharyngeal fibroma is an interesting condition, and rare in England, according to Martin[53]. The most urgent problem is one of treatment, and he reports on five cases.

The paper discusses nasopharyngeal fibroma under the headings of definition, incidence, etiology, pathology, clinical features and differential diagnosis as well as treatment, and is well worth reading in the original article.

The greatest danger of this tumor lies in its vascularity. This diminishes as the patient grows older, and complete regression occurs after the age of 25; however, treatment is usually necessary before this age is reached, and the author's cases were nearly all treated in their "teens". The various types of treatment are examined and these are: *1.* Injection of sclerosing solutions; *2.* Sex hormone therapy; *3.* Radiotherapy, and *4.* Surgery.

The author believes that surgery is the best treatment, and the transpalatal approach gives a good exposure of the tumor. He uses a transverse incision along the posterior edge of the hard palate and a second longitudinal incision about an inch long, joining the former at its central point. The posterior edges of the palate bone and septum are removed and a good view of the tumor is obtained. The mass is then removed as much as possible by diathermy.

If the tumor does not seem to be excessively vascular, and it is considered that most of it has been removed, the palate is re-sutured. Should there be any recurrence this may be dealt with by radiotherapy or diathermy, using post-nasal mirror control; however, if the tumor is very vascular and the operation apparently incomplete, a permanent opening or fenestation is left. This allows future inspection and treatment of the tumor by diathermy when necessary. The author has found this a very satisfactory method of treating nasopharyngeal fibroma. A permanent obturator is worn in the fenestration opening of the palate.

Handousa and associates[54] from Cario University discuss a clinicopathological study of 70 cases of nasopharyngeal fibroma. They claim the disease is not so common in Egypt as usually stated in the literature, and is found more frequently among the poor people.

An excellent pathological description is given with many illustrations of microscopic sections. Most of their cases

were between eight and twenty-two years of age and there were eleven females in the series, thus helping to disprove the view that this tumor is found only in males. The origin of the growth was always from an attachment above the pharyngeal opening of the Eustachian tube. Usually this was unilaterally placed in the roof of the nasopharynx, encroaching on the corresponding choana. This tumor may hang down in the nasopharynx and depress the soft palate; also, it may extend into the nose, compressing both the septum on one side and the sinus walls on the other.

The outstanding symptom is progressive nasal obstruction and recurring severe epistaxis. Headaches do occur, but pain is uncommon. The extension of the tumor may produce broadening of the nasal bridge, prominence of the cheek and eye with asymmetry of the face. Diminution of hearing on the affected side is noted, but suppurative otitis media is rare. A soft tissue X-ray will show the tumor hanging down in the nasopharynx and pharynx.

Treatment is discussed under the headings of conservative, irradiation and surgical excision. The ideal method of cure in the authors' experience is surgical excision. The main danger of the operation is hemorrhage, and correct pre-operative preparation such as transfusion is essential in the anemic patient. Satisfactory exposure is necessary, and the authors prefer a combination of the oral route with an external approach.

They feel the best possible external approach is that attained by Moore's lateral rhinotomy. The attachment of the tumor is located bimanually and the mucosa cut through by a suitable periosteal elevator. Dissection is then carried out by gauze, which is forced in between the base of the tumor and the underlying bone. As soon as the mass is freed it is removed by the oral route and the bed quickly packed with a long strip of vaseline gauze.

This method controls the profuse oozing which is liable to occur when the tumor is freed from the underlying structures, and in these cases pre-operative carotid ligature was not necessary. The operation is performed under general

anesthesia and is preceded by tracheotomy and pharyngeal packing. The nasopharyngeal packing is removed on the second day and the tracheotomy tube on the third day. Antibiotics are given for about a week and blood transfusions where necessary. Post-operative bleeding does not take place where the tumor has been completely removed. Recovery is rapid and a cure attained in three to four weeks.

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RESPIRATORY DISTURBANCES IN THE NEWBORN AND YOUNG INFANT.*†

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INTRODUCTION.

Conditions in the newborn and young infant producing respiratory disturbances with or without anoxia are many, and often of very complex nature. This discussion will in the main part be limited to those conditions in which abnormal structure or altered dynamics of the respiratory tract are responsible.

E. L. Potter[1] states that among 27,603 deaths occurring in the first 24 hours of life in the United States in 1946, 22,707 were due to prematurity. She believes "pulmonary immaturity," with inadequate oxygenation, was responsible in the majority of these cases. She goes on to state that between 1946 and 1949 death from anoxia in infants weighing more than 1,000 grams was 5.4 per thousand births at the Chicago Lying-In Hospital.

In 13,515 live births atelactasis was present 157 times, according to Clerf.[2] Ninety-five were in premature infants, while the remainder occurred in full term babies. Labate[3] prefers the term "anoxia." He states that this is one of the three major causes of death in infants; the other two being malformation and birth trauma.

THE NOSE, NASOPHARYNX AND PHARYNX.

Although subject to wide variations in size and shape, the nose is seldom abnormally developed. Anomalies of major importance in the nose are usually associated with other major defects, notably cyclops or cebocephalus. Obstruction of the nose in the newborn or young infant may produce severe asphyxial symptoms as well as difficulty in feeding. St. Clair

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Thomson[4] stated: "with rare exceptions all infants breathe entirely through the nose. If the nose of a sleeping infant is gently closed, he will waken and gasp for breath, instead of simply opening and using the mouth as an airway. Even when mouth breathing is partially adopted to supplement the nasal airway the latter remains the route preferred by instinct and as much air as possible is drawn through it. Mouth breathing is an acquired habit."

Persistence of the primitive nasopharyngeal membrane or congenital choanal atresia may produce asphyxia in the newborn infant. Beinfield,[5] and Mendovi and Beckmann[6] mention the occurrence of asphyxial symptoms in the newborn, due to the presence of atresia of the posterior nares. Mendovi and Beckmann report five cases of such atresia in the newborn. Bilateral occlusion was present in three cases, while in the remaining two the disease was unilateral.

Symptoms were most severe and occurred early in the bilateral cases, while in the unilateral cases asphyxia was not present or was of mild degree and appeared only during the feeding period. Presence of a unilateral nasal discharge was common to all the cases of unilateral atresia. Beinfield[7] reports a case of a 16-year-old girl in whom unilateral nasal discharge had been present from birth.

Early surgical treatment in the bilateral cases with asphyxial symptoms is urgent. The obstructing membranes should be perforated and a catheter left in place for a variable period of time post-operatively, in order to prevent the obstructions from re-forming. In the unilateral cases treatment may be deferred until the patient is older, when the more satisfactory transpalatine approach of Ruddy[8] may be employed. Choanal atresia is relatively uncommon, and most of the cases are reported in older children and adolescents. For this reason, many infants with this disease go undetected and probably have no symptoms from the disease until in later life.

Cysts and tumors of the nasopharynx may obstruct the upper airway and produce the same symptoms of asphyxia as congenital choanal atresia. Other anomalous tumors originating in the nasopharynx may be dependent and obstruct the larynx or lower airway, producing intermittent cyanosis. One

such case was recently treated by the author, and report follows:

Fig 1. Parasitic twin attached to the nasopharynx. A suture is holding the mass in the pharynx, preventing it from tamponading the larynx.

Case Report: A 4-week, white, female infant was seen because of attacks of cyanosis associated with a loud metallic, clicking sound on inspiration. The chest was clear on physical examination and X-rays were negative. During an attack the child would hyperextend her head. There was a loud clicking sound which could be traced directly to the larynx with inspiration. Marked inspiratory retraction and cyanosis developed. On further examination, a pedunculated mass resembling an accessory tongue was found hanging by a long pedicle from the nasopharynx into the larynx. This was drawn out and a suture placed through it to hold it in place in the mouth. The mass was smooth, somewhat flattened and had the appearance of a secondary, or accessory, tongue except for the absence of papillae.

At operation, the mass was removed by transfixing the stalk near the point of its origin and ligating it. The mass had its origin in the nasopharynx on the right lateral wall just below the torus. No other anomalies or defects were found. Following removal of the mass, the patient was entirely well, and when seen several months postoperatively was gaining weight and there had been no further attacks of cyanosis.

Examination of the mass revealed that it was covered with stratified squamous epithelium, or skin, and had most of the elements of skin, while the center was made up of loose connective tissue elements.

Since this structure has none of the elements commonly found in the region from which it arose, it must be assumed that it was a parasitic twin, or part of a second individual.

Edith Potter and others report the occurrence of parasitic twins attached in the pharynx and nasopharynx. In their cases, however, more parts of the second individual could be identified than in the present case.

As previously mentioned, infants are primarily nasal breathers. This is due to the relatively high position of the larynx, the more or less constant apposition of the tongue to the palate, and the relative shortness of the lower jaw. For this reason, space-consuming lesions of the pharynx apparently rarely produce obstruction, unless they are unusually large. Flaccidity of tongue may produce stridor when the infant is placed on its back. This is due, of course, to the pressure of the flaccid tongue against the posterior wall of the pharynx. Hypoplasia or subluxation of the mandible may cause glossoptosis, which may give rise to cyanosis and difficulty in nursing.

This condition may be fatal unless it is possible to overcome the obstruction of the pharynx caused by the position of the tongue. The condition of hypoplasia of the mandible, with obstruction due to glossoptosis, is known as Pierre-Robin syndrome. Cyst or lingual thyroid may produce similar obstructive symptoms requiring surgical relief.

LARYNX, TRACHEA AND BRONCHI.

The primitive larynx and trachea can first be identified as a local expansion in the upper anterior part of the primitive entodermal tube. A longitudinal constriction pinches off the anterior part of the expanded area, producing a short cylindrical appendage that communicates with the esophagus only in its uppermost part. This appendage branches into a right and left branch, the left giving rise to two divisions and the right to three. Clerf,[9] quoting H. E. Radasch, states that in embryos of the fifth week the laryngeal groove is bounded on each side by a rounded arytenoid swelling. Ventrally, at the base of the developing tongue, these join to form a transverse ridge over the third and fourth branchial arches. This is the epiglottic ridge, or fold. The arytenoid swellings and the epiglottic folds form a U-shaped ridge called the furcula. This slit-like opening may be termed the primitive glottis.

During the seventh week of development, each arytenoid swelling becomes bent or kinked near its midline so that the primitive glottis becomes T-shaped with the transverse portion at the epiglottis. At this time the arytenoid swellings

fuse and the primitive glottis is completely obliterated. Clerf considers this apparently a provision on the part of nature to prevent the passage of amniotic fluid down the respiratory tube during early stages of development.

About the tenth week, or shortly thereafter, this glottic fusion becomes dissolved and the arytenoid swellings lose contact with the epiglottis ventrally. The glottis then becomes oval in form. The position of the larynx in the neck of the infant is relatively higher than in the adult. It is at the level of the fourth cervical vertebra, while in the adult it has migrated down to the level of the sixth vertebra. This is in part due to the relative increase in the length of the neck.

Tucker[10] states that six millimeters is the normal subglottic AP diameter of the full term infant larynx. Five millimeters he considers as abnormal, while four millimeters indicates a definite stenosis. The larynx is also more flaccid and pliable than the adult larynx. Hill[11] believes that this is due to the fact that the glottic rim and the epiglottis are composed of yellow, elastic fibrocartilage rather than the white hyalin cartilage, which form the thyroid and cricoid cartilages.

Yellow cartilage is well known to be quite pliable, while hyalin cartilage is rigid and non-pliable. The pharyngeal aperture of the larynx in the infant faces dorsally at a fairly acute angle. After it has assumed the adult position farther down in the neck, the aperture becomes more transverse in relation to the long axis of the neck. In addition, the movements of the infant larynx are somewhat different than in the adult. In the infant, the arytenoids tend not only to move toward the midline but also ventrally toward the epiglottis, giving the impression of a distinct sphincter-like action to the glottic closure movement. In the adult the arytenoids do not move toward the epiglottis or ventrally but move to the midline.

Laryngeal stridor is one of the most common respiratory disturbances occurring in the newborn. It is characterized by noisy inspiration at birth or shortly thereafter. It is usually inspiratory, although when especially noticeable there may be an expiratory phase as well. Voice sounds are normal; cyanosis is rare, although there may be, as we shall see later, an

associated mild, diffuse atelectasis. In most cases the symptoms disappear after the second year, or even earlier.

Suffocation from this condition is rather rare, although fatalities have been reported. Two theories have been advanced to explain the mechanism of congenital laryngeal stridor, the first expounded by early authors who expressed the belief that the stridor was due to a central functional disorder, producing a disturbance in the coordination which takes place in ordinary breathing between the thoracic muscles on the one hand, and those of the larynx on the other.

Other writers, such as Hill and Tucker, express the belief that the stridor is due to the inspiratory collapse of the soft, pliable structures of the glottic rim of the infant larynx. Hill[11] concludes that the epiglottis, if short and flabby, might be sucked inward but feels that this would be secondary to the laryngeal rim in causing stridor. He states, however, that in one case traction on the epiglottis relieved the symptoms. From the author's experience, the epiglottis rim, rather than the epiglottis itself, by its flabbiness and infolding with inspiration is thought to be the cause.

Developmental defects of the glottis are chiefly of the web, band, or atresic type. Hypoplasia of the larynx is, of course, incompatible with life and extremely rare. It may be, according to Potter, associated with hypoplasia of the tongue, trachea, esophagus and lungs. Web of the larynx is probably the most common anomaly encountered, but even it is comparably rare. Hudson[12] states that between 1932 and 1949 only nine cases of laryngeal web had been diagnosed at Baby's Hospital, New York, where approximately 4,000 patients a year are admitted.

As previously indicated, the primitive epiglottic folds and epiglottis fuse primarily, and then this fusion later dissolves to establish the patency of the laryngeal airway; persistence of fusion of the arytenoids and epiglottis results in laryngeal web. The web usually occupies the space between the vocal cords near the anterior commissure; however, all degrees are recorded, varying from a slight web at the anterior commissure to almost complete fusion of the vocal cords, with only a small opening near the vocal processes or the posterior commissure.

Edith Potter reports three cases deserving special mention, in which she found identical malformations of the larynx. She states that there was a solid mass of cartilage approximately three millimeters thick extending across the entire larynx immediately below the vocal cords. The upper and lower surfaces were covered with normal epithelium. In two of these cases a hair-like canal connected the upper and lower segments and was present posteriorly in the midline. These findings may be explained by the views of Arey,[13] who states that the larynx develops somewhat differently in its upper and lower parts. He feels that the lower portion forms around the stem of the trachea, whereas the upper part, including the vocal folds, arises from the pharyngeal floor in the region of the primitive glottis.

Symptoms produced by congenital laryngeal web vary considerably in severity, depending upon the degree of obstruction present. Thus a small anterior web may produce only hoarseness. With a greater degree of glottic occlusion stridor and dyspnea appear, while with even more severe degrees of obstruction, cyanosis and retraction of the chest wall make their appearance.

Flaccidity of the subglottic mucous membrane, lack of rigidity of the cricoid cartilage or its actual absence may also be responsible for stridor, dyspnea and cyanosis. These conditions, however, are uncommon, and are not present as often as congenital web.

Obstruction in the trachea can be divided into three major categories: *1.* Compression due to extrinsic lesions; *2.* Intrinsic obstructions, and *3.* Lack of rigidity of the tracheal wall. Tracheal tumors are rare, but when present, according to Potter, are most often found in the upper third of the trachea. They are principally papillomas, fibromas and angiomas. Symptoms produced are commonly a brassy cough and associated respiratory difficulty.

Collapse of the trachea is rarely responsible for respiratory difficulty in newborn infants. In the normal infant, a certain degree of inspiratory narrowing of the tracheal lumen is almost always present and by external pressure the trachea, and especially the bronchi, can be easily collapsed. Unusual

flaccidity or absence of the tracheal rings may be responsible for symptoms of obstruction and stridor.

Inspiratory collapse of the tracheoesophageal wall may produce dyspnea and cyanosis, thus Phelps[14] reports a case of a newborn infant who was a blue baby at birth and suffered from attacks of cyanosis at intervals. On examination, this child had a normal larynx. The trachea was observed to close on inspiration from collapse of the party wall. No other abnormality was found. The infant lived a few days, and at autopsy showed a normal larynx. Phelps states that very few tracheal rings were present and that these were smaller than normal. This, he felt, accounted for the tracheal collapse. The author himself has observed one case of tracheal obstruction due to collapse of the party wall. This infant was several days old when examined; was bronchoscoped without difficulty, and on inspiration a segment of party wall just above the carina was observed to balloon into the trachea, producing a loud, fluttering sound. This child survived; stridor gradually subsided and had cleared entirely in six months.

Tracheobronchial compression may be produced by congenital cardiovascular defects. Hollinger,[15] and later, Baker,[16] divided these into two major groups. The most common are due to congenital cardiac defects. In these cases, compression of the left major bronchus is produced when an enlarged left auricle or ventricle encroaches on it. In the second group, anomalies of the aorta and great vessels produce constriction of the trachea and/or the left major bronchus. Double aorta, or so-called vascular ring, is the most common anomaly of this group. The aorta divides and passes to either side of the trachea, reuniting between the trachea and esophagus. Sometimes it unites again behind the esophagus. The small size and softness of the infant tracheobronchial tree allows it to be compressed easily. Obstructive emphysema, as well as atelectasis, may result. These defects, can, in some instances, be corrected by the thoracic surgeon. Early diagnosis and treatment are essential, because the prolonged use of an indwelling tracheotomy tube to hold open the collapsed trachea may produce erosion and fatal hemorrhage.

Congenital mediastinal cyst is also sometimes responsible

for compression of the trachea. Obstruction is usually at, or above, the carina.

So-called enlarged thymus has been incriminated as a cause for tracheal compression; the author, however, does not hold to this view. Edith Potter states. "To my knowledge, hyperplasia of thymus has never been proved to have been the cause of death in the neonatal period. I do not believe it causes death, either by mechanical compression of the trachea or by inducing some occult disturbance, generally designated as thymicolymphaticus. Nor is there any reason to believe that it causes stridor or asthma. The fact that the function of the thymus gland is unknown seems to be largely responsible for its indictment as a cause of sudden death. Years ago, when the small thymus of a sick infant was believed to be the normal size, the discovery of the non-atrophic and normal thymus in any infant who had been ill led to the assumption that the gland was hypertrophied. Large-scale X-raying of the chest to determine thymic size was done, and many diagnoses of enlarged thymus were made. Since all lymphoid tissue shrinks under X-ray therapy the recommendation was made that the gland be reduced in size by this means. In many communities the diagnosis is still made and X-ray therapy given."

She goes on to state: "Among the 8,000 autopsies on infants under one year of age that I have been fortunate enough to observe, the thymus has never shown any abnormality that might have led to death. It cannot be denied that infants occasionally die unexpectedly, but there is no evidence, unless an actual tumor is present, that the thymus is responsible or that X-ray treatment of the thymus would have prevented death."

Tracheoesophageal fistula is rare but responsible for severe dyspnea, cyanosis, and death in young infants. Symptoms usually appear at birth, are more severe during the feeding period and result from aspiration when feeding is attempted. This anomaly may vary from an opening or fistulous communication between the trachea and esophagus to complete absence of the upper segment of the esophagus. In the latter case, the upper end of the esophagus ends in a blind pouch while the lower segment is attached to the lower end of the trachea communicating with it through a single opening. This

condition is thought to be the result of the incomplete separation of the primitive larynx and trachea from the primitive entodermal tube.

Case Report: A full term male infant with marked cyanosis, especially after attempted feeding. Bronchoscopy performed, and an anomalous opening into the lower segment of the esophagus was observed between the right and left main stem bronchi just at the carina. Bronchoscopic aspiration was performed several times during the first 24 hours of life, with temporary relief of dyspnea and cyanosis. The defect was repaired and the patient survived for three months postoperatively. Pericarditis was found at autopsy and was probably the cause of death. The anastamosis of the lower segment with the blind pharyngeal pouch was successful and had healed completely. (Case of Frank L. Gerbode, M.D.).

THE LUNGS.

Disturbed pulmonary function is by far the greatest factor in the production of asphyxia in the newborn and young infant. As previously indicated, atelectasis is one of the major causes of death in the neonatal period. Knowledge of the changes in the lung, both anatomically and physiologically is prerequisite to an understanding of the various pathological states of the lung which occur at birth.

Until recently, it has been thought that the initial expansion of the human infant's lung was a very rapid process, completed in a very short period of time; however, evidence has been accumulated to show that this process is a gradual, uneven procedure requiring hours, or even days in some cases, to complete. Thus Farber and Wilson[17] have stated that an infant's first breath is his most difficult one, and they found that it takes a pressure equal to about 25 cm. of water to expand the infant's lung.

Clement Smith[18] states that a full term infant can exert a pressure of from 40-50 cm. of water on his lungs. He feels that this is the upper limit of safety for pressures applied to it. The infant's cry at birth with the consequent closure of the glottis and spasmodic increase in intrathoracic pressure undoubtedly plays a part in this initial process.

Hilding[19] and others have found roentgenographic signs of atelectasis persisting up to, and in some instances beyond, 24 hours in otherwise perfectly healthy newborn infants. Martin and Friedell[20] point out that the cardiac silhouette changes markedly with atelectasis in the newborn. They point out

that the transverse diameter of the heart and cardiothoracic ratio are increased in atelectasis; also of significance is the fact that the superior mediastinal shadow often appears wider. These changes are, of course, much more marked than those normally observed in an infant during phases of inspiration and expiration.

The diaphragm does most of the work in pulling the lung open. Hilding also believes that this, as well as the change from the curled-up to the opisthotonic, stretched-out position, exerts the necessary piston-like pull that opens the lung initially. Few references occur in the literature regarding the thoracic cage and its role in the expansion of the lung. The writer believes that the state of the thoracic cage is extremely important in this initial expansion process. He will attempt to elucidate this problem further and justify his position; however, at this point it is necessary to digress and consider certain other questions related to the expansion process.

Respiratory movement in utero have been observed by many, and there is evidence to suggest that respiratory movements in utero occur in the human infant. Clement Smith, however, cautions that present knowledge concerning the development of thoracic movements in utero does not allow the assumption that the onset of breathing at the time of birth is merely a continuation of this fetal activity. There is contradictory evidence and difference of opinion regarding the presence or absence of amniotic fluid in the fetal respiratory tract. Labate states that it probably fills the trachea but not the alveoli.

Gruenwald[21] found fluid only in the alveoli of stillborn infants. He found also that fluid would expand the atelectatic lung with far less pressure than gas; but, on the other hand, Hilding failed to demonstrate the presence of amniotic fluid in the respiratory tract of experimental animals, in which he had tied off the trachea after observing fetal respiratory movements in utero. The author has bronchoscoped infants with "fetal atelectasis" and has not found convincing evidence of the presence of amniotic fluid.

From the above evidence, and other evidence to be presented later, the writer believes that fetal respiratory move-

ments are of such a nature as to allow the development of the thoracic muscles without the exchange of fluid or change in the volume of the thorax, and believes that this condition exists in the human fetus.

Other factors are responsible for the initiation of the rhythmic respiratory cycle. According to Clement Smith, the externally applied stimulus brought about by contact with a new environment and the chemical stimulus of hypoxia produced by separation from the maternal circulation produce this rhythmic respiratory cycle. Modifying this picture is, of course, the state of the central nervous system. One sees the gasping, irregular type of respiration more frequently in the premature infant. Smith goes on to state that high concentrations of oxygen rather than carbon dioxide tend to restore the higher centers and thus rhythmic respiration.

It must be remembered that the fetus has an extraordinary tolerance for anoxia. This is in part due to certain qualitative differences in fetal and adult hemoglobin. The former has a greater affinity for oxygen at ordinary pressures, yet dissociation readily occurs at tissue pressures. The blood of a full term infant contains a mixture of adult and fetal hemoglobin. The premature infant, on the other hand, has a greater percentage of the fetal type hemoglobin. This would perhaps tend to make the premature infant more resistant to anoxia than the fully developed infant at term. Higgins[22] observed a human fetus inside the intact membranes making vigorous respiratory movements for fully 28 minutes after the ovum had been separated from the maternal circulation.

Brandau[23] records a remarkable experience. He was called to attend a woman who had delivered a six months, premature infant with membranes and placenta intact. Forty-five minutes after delivery the "bag of waters" was ruptured and the baby recovered from the enveloping membranes. Brandau says that the child's color was good, heart action excellent, and that he had no difficulty in getting the baby to breathe, after clamping the umbilical cord; however, death occurred 24 hours later because of respiratory difficulty and prematurity. Autopsy was not obtained. Brandau explains his observations by assuming that the intact fetal placenta was able

to absorb enough oxygen from the atmosphere for the needs of the fetus. No respiratory movements were seen through the intact membrane.

Important also in the initial expansion process in the newborn lung is its state of development. Edith Potter believes that the vascular bed, if underdeveloped, is responsible in many cases for atelectasis occurring in the neonatal period. Farber and Wilson[24] were able to differentiate between the unexpanded infant lung and the one which has expanded and then becomes atelectatic. In the former, the cells lining the alveoli are of a high cuboidal shape and quite regular. With the initial expansion process these cuboidal cells become stretched out flat and irregularly spaced. Consequently, if atelectasis occurs after this initial expansion the alveoli have a slit-like appearance, the cells are flattened, irregularly spaced and the vascular bed may be engorged. This suggests the possibility that closure of the foramen ovali and ductus arteriosus which shunt the blood through the pulmonary artery and consequently through the lungs, may play an important part in the initial expansion process.

Miller and Jennison,[25] Potter, Behole, Gibson and Miller,[26] and others have described a syndrome which is best termed resorption atelectasis with hyalin membrane. This disease occurs more commonly in premature infants and those delivered by Ceasarean section. Clinically the infant breathes spontaneously and seems perfectly normal to percussion and auscultation, and then within a few hours becomes cyanotic. The initial cyanosis may be relieved by oxygen, but the respiratory rate and effort become more pronounced. There is increasing retraction of the ribs and sternum with each respiration. At this time signs of massive atelectasis are usually present. The cyanosis becomes more severe, the respiratory effort more pronounced and is no longer relieved by oxygen. Death frequently occurs within the first two days.

On autopsy the the lungs are characteristic; they are the size of well-expanded lungs but have the consistency of liver. They are of uniform dark red or purple color. Pathologically, there is wide-spread collapse of the alveoli and the alveolar ducts. There is capillary engorgement. The inner surfaces

of some of the alveolar ducts that remain open are covered by an irregular layer of homogenous and acidophilic material which may be the result of anoxic necrosis of the lining epithelium. Miller, Potter and others believe that the presence of this hyalin material blocks the exchange of respiratory gases.

Labate is of the opinion that pulmonary hyalin membranes are formed as a result of the absorption of the fluid portion of the aspirated amniotic sac contents. Ranstrom,[27] after analyzing 30 cases of newborn infants with hyalin membranes in the lungs, concludes that the membranes are of no significant functional importance in the vast majority of these cases. He thinks that the presence of large areas of atelectasis are responsible rather than the presence of a few scattered areas of hyalin membrane.

Farber and Wilson have been able to produce experimentally hyalin membranes by hyperventilating the excised lung and introducing a foreign substance, such as ink or exudate, into the trachea. Bubbles of air become trapped in the alveoli, thus forcing the hyalin substance against the alveolar wall and producing, according to these authors, the typical picture. They also found hyalin membranes in other diseases where extreme respiratory efforts were made because of cyanosis. Of further significance is the observation of Behole, Gibson and Miller, who found that these lungs could be made to expand by exerting sufficient negative pressure on the lung surface.

The author has been fortunate in being able to collect 11 cases which clinically fit into the picture described above. Six of these cases were examined and treated by the author, while the remainder were treated by other physicians. Marked inspiratory retraction of the sternum was present in all cases and increased in severity until traction was applied to the sternum. Cyanosis was well developed and appeared after a short period of well-being following delivery, in all cases except one. In all cases recovery was prompt and permanent. Traction was maintained on the average for 24 to 48 hours. The longest period of time for which it was maintained was five days. All cases treated were considered in the premature

group; the largest weighing five pounds, seven ounces, and the three smallest infants weighing three pounds or slightly less. The details of two of these cases will be presented since they are complete and X-rays were taken before, during and after treatment.

Case Report: A four and one-half pound female premature infant exhibited sternal retraction at birth. This increased and cyanosis was first noticed at the end of 12 hours. Bronchoscopy was performed and the tracheobronchial tree found to be clear. There was no improvement following this procedure. Sternal traction was applied and immediate improvement in the child's color was noted. On the following day breath

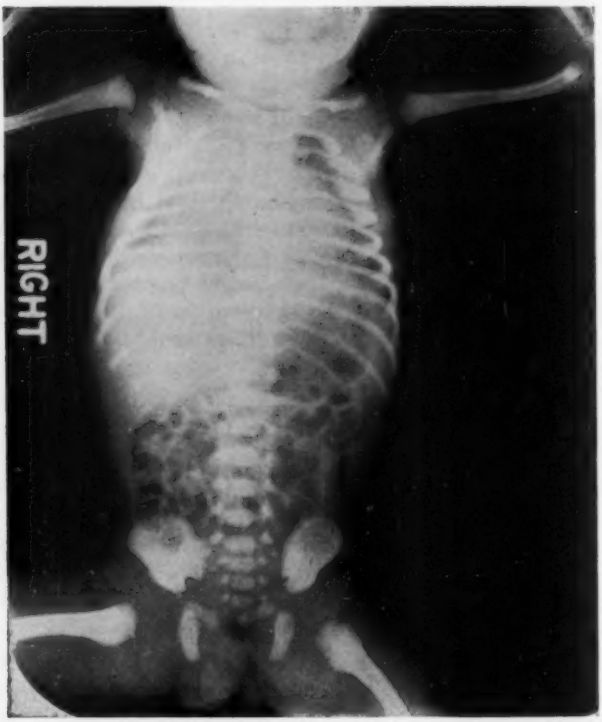

Fig. 2A. Anterior-posterior view of the chest before the application of traction. (Case of Dr. Ralph Fisher)

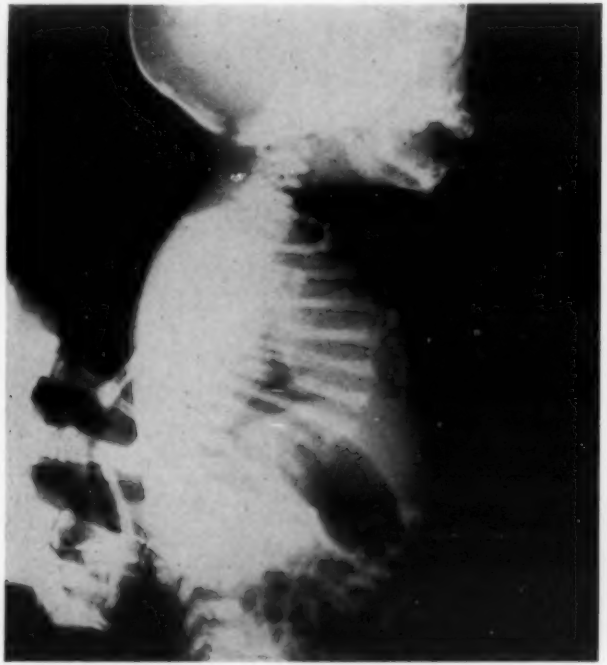

Fig. 2B. Lateral view of the chest before the application of traction—expiration.

Fig. 2C. Lateral view of the chest before the application of traction—inspiration.

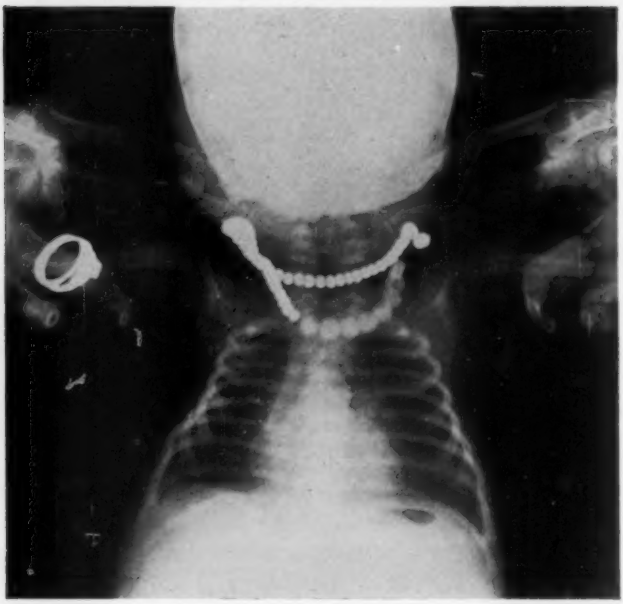

Fig. 2D. Anterior-posterior view: Complete clearing. Note changed appearance of the cardiac silhouette.

sounds were heard throughout the chest and the color was good. The suture was removed from the sternum and the patient had no further respiratory difficulty.

In the following case the author was extremely fortunate in being able to secure films taken before, during and after the application of sternal traction. These films were taken as nearly as possible in full expiration and inspiration.

Case Report: A five-pound, two-ounce male infant born by Ceasarean section. Cry and color were described as excellent immediately following delivery. Even at that time, however, retraction of the sternum was noted. Two to three hours later the infant became cyanotic. He responded to oxygen, but in spite of this therapy the extremities remained dusky. Two days after delivery traction was applied, with almost immediate improvement in color. Traction was left in place for approximately 24 hours. Subsequently the chest remained out and the patient is alive and well today.

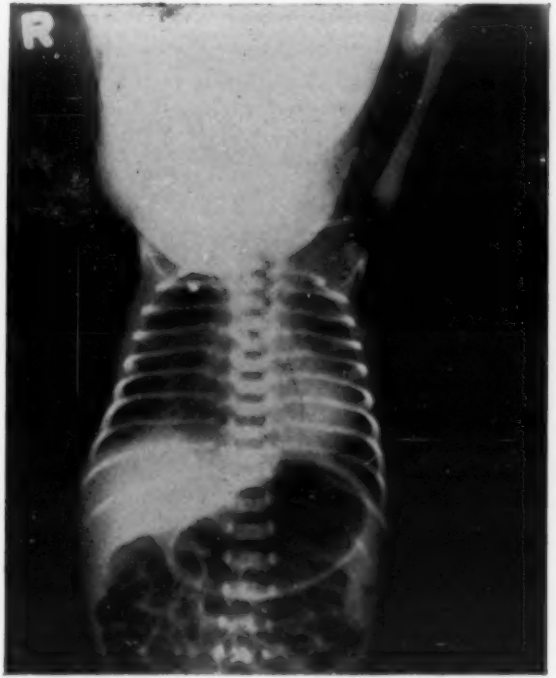

Fig. 3A. Anterior-posterior view of the chest before traction. (Note the air containing tracheobronchial tree which can be traced to its finest divisions).

Fig. 3B. Lateral view—before treatment—inspiration.

Fig. 3C. Lateral view before treatment—expiration.

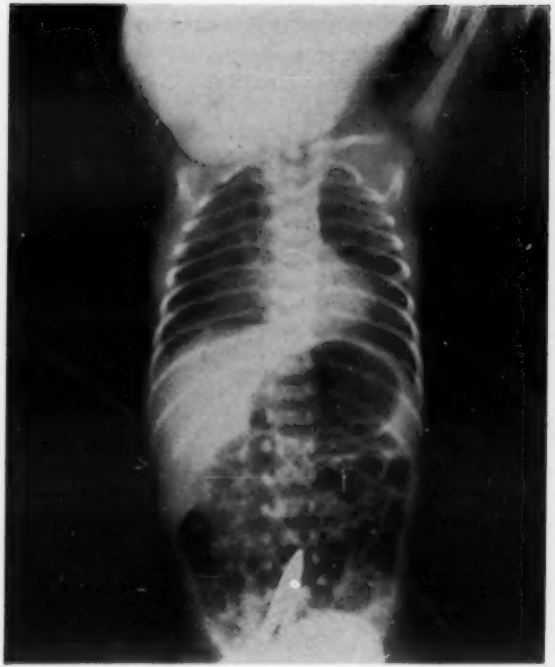

Fig. 3D. Anterior-posterior view of chest 12 hours later with traction applied.

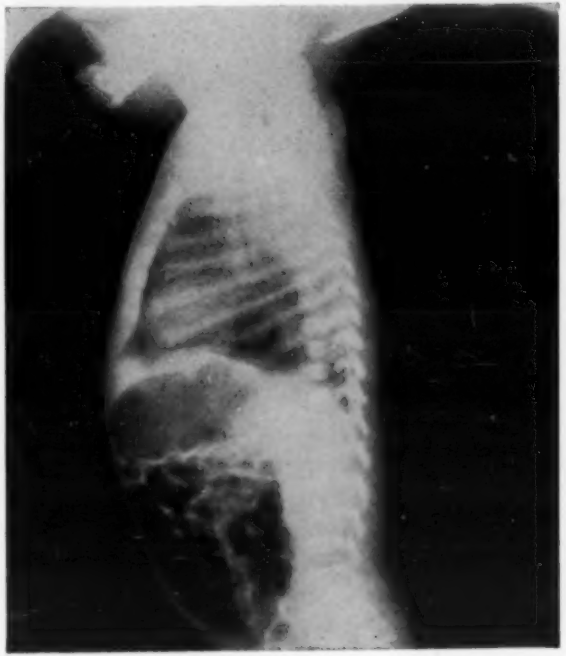

Fig. 3E. Same as (D)—lateral view.

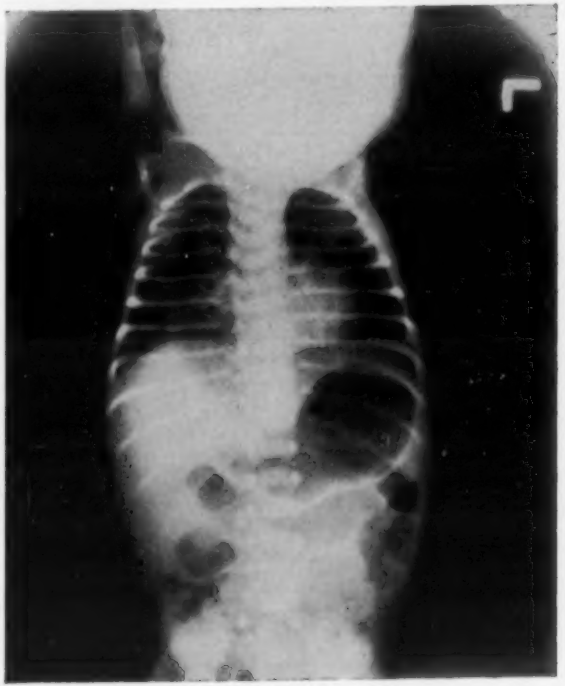

Fig. 3F. Anterior-posterior view — 48 hours after removal of traction.

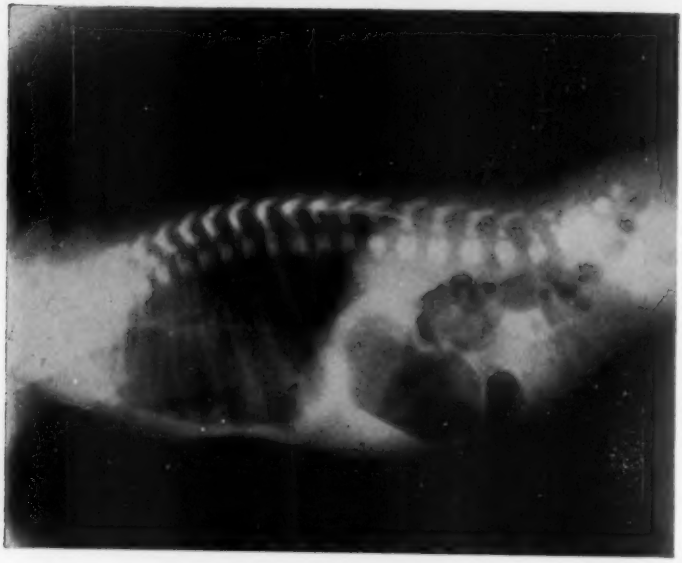

Fig. 3H. Same as (G)—Inspiration. (Note slight dorsal movement of the sternum).

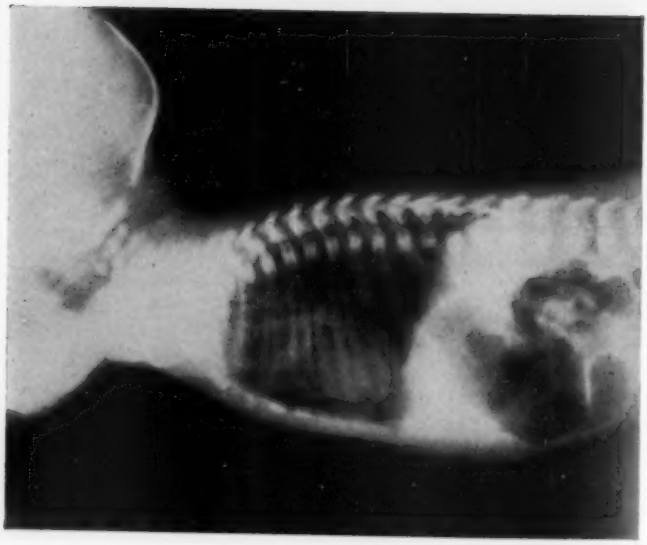

Fig. 3G. Lateral view—48 hours after removal of traction.

ELEVEN CASES OF ATELECTASIS IN THE NEWBORN TREATED WITH STERNUM TRACTION

Delivery	Sex	Weight	Cyanosis	Thorax	Chest X-ray	Remarks
1. Spontaneous	M	4 lbs. 7 oz.	Two hours after delivery, marked	Marked retraction	Bilateral patchy atelectasis; clear after traction (4 days)	Traction applied 18 hours after delivery. Immediate improvement in color. Traction removed at 4 days; sternum remained out.
2. Caesarean	F	5 lbs. 2 oz.	Cyanotic shortly after delivery	Retracted from birth	Bilateral patchy "fetal atelectasis," showed marked X-ray improvement with traction	Color improved with traction; traction applied 12 hours; chest remained expanded.
3. Breech	F	2½ lbs.	Color labile	Marked retraction, rapid respiratory rate	None	Traction applied with improvement. Respiratory rate slowed. Traction in place 4 days and released; patient well 1 week, retracted again. Traction re-applied with improvement After 5 days no retraction; alive and well 6 months later.
4. Caesarean	F	4 lbs. 3 oz.	Marked from birth	Retracted from birth	None	Placed in airlock at delivery; became worse. Traction applied 30 hours after delivery; immediate improvement in color. No cyanosis in room atmosphere; traction for 12 hours. Complete recovery.
5. Caesarean	M	4 lbs. 8 oz.	1½ hours after delivery	Retracted 1½ hours after delivery		Placed in Bloxom airlock; condition worsened with increase in cyanosis and retraction. At 20 hours traction instituted with improvement in color and respiration. Traction removed 8 hours later; no cyanosis.
6. Spontaneous	F	4 lbs. 6 oz.	Marked at 12 hours	Retracted from birth	Marked bilateral "fetal atelectasis;" cleared after traction	Bronchoscoped; no improvement. Traction applied with marked improvement. Removed after 24 hours with no further difficulty.

ELEVEN CASES OF ATELECTASIS IN THE NEWBORN TREATED WITH STERNAL TRACTION

Delivery	Sex	Weight	Cyanosis	Thorax	Chest X-ray	Remarks
7. 7½ mos. Premature	Not stated	Approx. 3 lbs.	Marked at 3 days, in spite of 0_2	Retracted at 3 days	None	Traction for 48 hours. Immediate and marked improvement, which was sustained. (Case of Dr. George Y. Elson).
8. Not stated	Not stated	Approx. 3 lbs.	Marked at 3 days, in spite of 0_2	Retraction marked at 3 days	None	Marked immediate and sustained improvement after 48 hours of traction. (Dr. Geo. Elson)
9. Breech, very difficult	M (Negro)	5 lbs. 7 oz.	Marked, incompletely relieved by 0_2	Extreme retraction at birth		Immediate improvement in respirations and color with traction. Recovered in 24 hours without retraction or distress. Convulsions at 5 days; E Coli meningitis cleared with antibiotic therapy. Well at one month. (Case of Dr. Benson Roe).
10. Normal spontaneous	M (Negro)	4 lbs. 13 oz.	Present at 5 hours, relieved by 0_2; on 3rd day unrelieved by 0_2	Retraction at 5 hours	None	Traction on the 3rd day; suture pulled out of sternum several times. Slight but definite improvement. Septicemia hem. staph. sur. due to sternal wound infection. Cleared with penicillin; baby discharged well. (Case of Dr. Benson Roe).
11. Spontaneous Breech (Pre-eclamptic mother; first of twins)	M	6 lbs. 3 oz.	Absent at birth; present 2 hours after delivery. Relieved by 0_2. Unrelieved at 24 hours	Retraction from birth	None	Traction (suture through the xyphoid) at 30 hours. Cyanosis disappeared immediately. Traction removed after 3 days. Full recovery. (Case of Dr. Benson Roe).

NOTE: The author's six cases treated by sternal traction were done in the following manner: A braided cotton or silk suture was passed through the xyphoid. The suture was then attached to several stout rubber bands which were in turn attached to the incubator roof. Tension was adjusted until the desired effect was obtained and varies from one case to the next.

The above experience suggests the distinct possibility of some other explanation for the syndrome termed "resorption atelectasis with hyalin membrane." Although statistical evidence is lacking, and the series of cases is admittedly small, the writer believes that serious consideration should be given to another theory of the pathogenesis of this condition, namely the theory of "congenital flail chest." Traumatic flail chest is too well-known to need discussion in this paper. The mechanism in the above cases, the author believes, present certain striking similarities to the traumatic type. Most important of these is the paradoxical movement of the sternum, which retracts with inspiration in both types of "flail chest."

In the newborn and especially in the premature infant, the thoracic cage is a relatively soft and somewhat pliable structure. The lungs, on the other hand, are elastic and tend to resist the pull of the diaphragm, which even at this stage of development is a comparatively powerful muscle. In this manner the chest wall, the least resistant of the two, gives way. When traction is applied to the sternum, the diaphragm is given something to fix against and more of this force is directed toward "pulling" open the lung.

This phenomenon was strikingly illustrated in one of the above cases. In this instance the infant's sternum began to retract and cyanosis returned as soon as traction was released. When the sternum was again stabilized cyanosis disappeared immediately and good oxygenation was maintained even in room atmosphere. This observation was repeated several times.

It must be emphasized here that traction must be resilient to allow excursions of the chest wall, and of sufficient force to counterbalance the pull of the diaphragm. Further support for this theory can perhaps be derived from the observations of Berglund and Eder.[28] These workers performed open thoracotomy on two infants with this syndrome. They found that the lungs inflated quite readily with the chest open, when positive pressures were used which had previously failed to inflate the lungs with the chest wall intact. Their work tends to confirm the observations of Flagg,[29] who found that he could inflate the atelectatic lungs (of infants) under water,

with pressures which would not inflate them *in situ*. In Berglund and Eder's two cases, the lung protruded through the thoracotomy wound. This would suggest that the fully expanded lung tended to occupy more volume than the thoracic cavity provided. This might have been due to the production of emphysema, but this does not seem likely in view of the low positive pressures used for inflation.

Baggott[30] observes that in massive atelectasis something must make up for the lost volume of air in the chest. He suggests that this lost volume of air is replaced by blood in the form of intense capillary engorgement of the pulmonary bed. He states the "law of the thorax" thusly:

"The mass of the alveoli, of the vessels (or of the blood) or of the interstitial tissues may vary, but the sum of their masses must be at least equal to the capacity of the thorax in maximal expiration."

Baggott suggests that before birth the lungs fill the chest because of the presence of a supplemental mass of blood in the lungs. The observations of Farber and Wilson do not support this view; also, this would not explain the changes in the cardiovascular system which shunt the blood through the lungs for the first time at birth. Another possibility suggests itself. Collapse of the sternum may allow reduction of thoracic volume in utero, and the paradoxical motion may permit the development of intrauterine respiratory movements, without affecting the volume of the chest. This offers a possible explanation for paradoxical movement of the sternum, observed so frequently in newborn infants; thus, "congenital flail chest" may represent a fetal type of respiratory movement. The writer realizes that there are no observations of direct nature at the present time to confirm this theory.

In the preceding paragraphs the writer has discussed collapse of the lungs, which he believes is due to pressure changes exerted at the lung surface. Congenital defects of the diaphragm, with herniation of the abdominal contents into the thorax, by consuming the available space prevent the expansion of the lung. These defects may produce severe respiratory distress and marked cyanosis. Resuscitation is, as a rule, only temporary, and these distressed infants rarely survive

beyond the 24-hour period. The defect is usually not found until autopsy, and the diagnosis will not be made unless one is thinking of this defect. Surgical reduction and repair of the defect in the diaphragm must be done early.

The following is an illustrative case in which the author was requested to bronchoscope a patient who was apparently suffering from unilateral massive atelectasis:

Case Report: A full term male infant whose delivery was uncomplicated. Cyanosis was present from birth and was quite marked unless oxygen was given. The respirations were vigorous and rapid. The chest wall did not retract. The left chest was entirely flat to percussion and no breath sounds were heard. The heart was heard and felt on the right side of the chest. The abdomen was flat and the costal margins were seen to draw in slightly with each inspiration. No bowel sounds were heard in the chest. Because of the flatness of the abdomen and the absence of sounds in the left chest, together with displacement of the heart to the right side, congenital diaphragmatic hernia was suspected. X-rays confirmed this opinion.

Dr. Henry Silvani operated on this patient and found a large defect in the left leaf of the diaphragm, through which most of the bowel and stomach had herniated. The hernia filled the entire left chest and was easily reduced. The left lung was found compressed against the mediastinum and completely collapsed. The diaphragmatic defect was closed and a small incisional hernia made. The hernia contents could not be immediately replaced in the abdomen because the capacity of the abdomen was too small to allow closure of the diaphragm without tension.

Approximately ten days later the hernia was repaired. Serial X-rays of the chest showed that the left lung gradually expanded, and the last film showed complete expansion. The child made an uneventful recovery except for an episode of bowel obstruction, necessitating a second laparotomy. Following this experience the child was entirely well and developed normally. When last heard from, six months later, he was entirely well.

Until fairly recently congenital diaphragmatic hernia was thought to be inoperable. Harper and Anderson[31] in 1930 wrote: "The heart and lungs are displaced to the opposite side of the invading abdominal organs. There is also a marked lack of development of one or both lungs, a significant point to be considered in any proposed surgical treatment." They concluded that immediate surgery is contraindicated.

As previously indicated, incomplete development may prevent the expansion of the lung. Arrest of development may occur at any stage and may be slight or extensive. Symptomatology is quite variable. No symptoms may be present or severe respiratory distress with cyanosis may occur. In the latter case the outcome is usually fatal. Bronchoscopy and bronchography may be of great value in differentiating this

from other conditions, as the following case illustrates. It is also of interest to note that the right lung is most often affected. No explanation for this is offered. Death may occur suddenly at any time without warning.

Case Report: A one-month-old female infant had been wheezing from birth, but at no time had exhibited respiratory distress or cyanosis. She was referred for bronchoscopy because of the appearance of the chest X-ray. The right side presented an almost opaque appearance. The cardiac silhouette was absent from the left side. Bronchoscopy was performed and revealed that the right main bronchus ended in a blind sac. Several tiny openings in it could be made out. Bronchography revealed a small cystic dilatation of the end of the right main bronchus. The left main bronchus was tremendously elongated and revealed that the left lung had herniated around to the right side and had almost completely filled the right thorax as well as the left. The patient expired suddenly one month later, and no explanation for her death was found at autopsy, which confirmed the above findings in the chest. No other associated anomalies were present; however, the head in this case unfortunately was not examined.

Thomas and Boyden[32] report three cases of agenesis of the right lung, including one death while a foreign body was being extracted from the left main bronchus. It is of interest to note that the right side again was the side of predilection for the failure of development. These authors present an extremely extensive review of the literature on this subject and suggest that lack of Vitamin A in the maternal diet or an hereditary factor may be responsible.

Two types of atelectasis in the newborn have been discussed in the preceding paragraphs, *i.e., 1.* Collapse due to pressure changes exerted at the lung surface, and *2.* Failure of pulmonary tissue to expand. A third type, due to the resorption of air from a segment of pulmonary tissue distal to an obstructed bronchus is equally important. Bronchoscopy is most successful in this type of case since it is usually due to aspiration. Obstructing material may find its way into the tracheobronchial tree at any time during the delivery or postnatal period.

Farber and Wilson, Potter, Labate and others mention the presence of large numbers of epithelial cells which are occasionally found filling the alveoli of the lungs of infants dying of asphyxia. This phenomenon is thought to be due to the initiation of pulmonary respiration brought on by a period of intrauterine anoxia. Likewise so-called "congenital pneumonia" is thought to be the result of intrauterine aspiration

of infected amniotic sac contents. The writer has had no experience with either of these conditions.

Atelectasis following postnatal aspiration is easily understood since the mechanism of its production and the pathology are similar to the condition in the adult. One or both sides may be involved, or it may be limited to one or two lobes of the lung. The amount of distress produced depends upon the extent of involved pulmonary tissue. It may subside spontaneously or result in pneumonia. Aspiration atelectasis in the newborn should not be confused, however, with "persistent physiological fetal atelectasis." In the writer's opinion any infant who shows persistent areas of collapse which fail to expand after a reasonable period of observation should be bronchoscoped.

The following case illustrates the sequence of events which may result from aspiration during feeding:

Case Report: A nine pound, eleven ounce white male infant delivered spontaneously. Color was excellent and cry vigorous. At the time of the breast feeding the infant was noticed to be cyanotic and apneic. The baby responded to Carbogen and ½ cc. of Coramine. Forty-eight hours later chest films revealed the presence of atelectasis in the right upper lobe. By this time the baby was running a low-grade fever and the X-ray findings of atelectasis persisted. Fever and prostration continued for about a week and responded to penicillin. Two months later the baby was referred because of persistent consolidation of the right upper lobe. X-rays taken immediately before bronchoscopy confirmed this finding. At bronchoscopy the tracheobronchial tree was entirely clear; however ¼ per cent Neosynephrine was applied topically to the orifice of the right upper lobe and spot suction applied. Films were taken following bronchoscopy showing partial clearing, and on the following day the right upper lobe was entirely clear. The baby has developed normally and at last report, some three months later, was entirely well.

Diffuse bilateral partial atelectasis may occur with partial obstruction of the upper airway as the following case of laryngeal stridor associated with atelectasis illustrates:

Case Report: A three-month-old white male infant who had had attacks of stridor, dypsnea and cyanosis from birth was referred because the chest X-ray revealed a wide upper mediastinal shadow with considerable collapse of the left upper lobe and patchy areas of density in the remaining portions of the lung. When examined under anesthesia the larynx was found to be flaccid and the aryepiglottic folds collapsed inward with each inspiration. Bronchoscopy was normal although small amounts of mucous were aspirated. Following this procedure the mediastinal shadow disappeared and the cardiac silhouette assumed the adult configuration. Several days later the patient had another attack of cyanosis and a chest film revealed essentially the same findings as before bronchoscopy.

SUMMARY.

Atelectasis is the most frequent finding in neonatal death. Birth trauma and congenital defects are of lesser importance.

Defects of structure and function of the upper air passages producing respiratory disturbances are discussed. A case in which a dependent congenital tumor attached to the nasopharynx, producing glottic obstruction, is presented. The tumor is believed to be a so-called "parasitic twin."

Conditions giving rise to tracheal obstruction in the infant are reviewed. Three types may be recognized: intrinsic, usually produced by tumors, and more rarely congenital malformation; extrinsic lesions producing compression, commonly due to congenital cardiovascular defects, and rarely mediastinal cyst or tumor; and collapse of the tracheal wall, associated with absent or defective cartilaginous tracheal rings. The writer's experience with collapse of the tracheoesophageal wall in one case is presented. Contrary to earlier beliefs, massive diaphragmatic hernia is best treated by early surgical reduction and closure of the defect. A case report is included.

Repeated, early bronchoscopic aspiration in tracheo-esophageal fistula may be life-saving and make surgical repair possible. A case report is included.

The syndrome "pulmonary hyalin membrane with resorption atelectasis" is discussed. Eleven cases treated by sternal traction are presented. It is suggested that according to the "law of the thorax" the paradoxical movement of the chest wall allows intra-uterine respiratory movements without changing the volume of the thorax appreciably. It is further suggested that the movements of the thorax observed in "congenital flail chest" are persistent fetal respiratory movements. Observations of a direct nature in support of this theory are not presently available.

Agenesis, or failure of pulmonary development, is variable in extent and degree. Symptomatology is, therefore, varied. This anomaly is a rare occurrence and most frequently involves the right lung. A case report is included.

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EXTERNAL UMBO BLEBS.

Non-Inflammatory Lipoid Vesicles of the Tympanic Membrane.*†

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Myringitis bullosa is well-known to all who practice otology, and yet the literature on this subject is suprisingly meager. Even less has been written concerning non-inflammatory blebs commonly seen at the umbo. The purpose of this paper is to record observations on lipoid blebs at the umbo, an entity that has been almost completely ignored. The material presented was obtained in the course of exactly 24,000 consecutive and unselected office examinations during the five-year period 1949-1953.

My attention was first directed to blebs at the umbo in 1946, when a 2 mm. glistening elevation was observed at the tip of the malleus handle (see Fig. 1). The patient had no history of ear disease and no complaint referable to the ear. Incision into the bleb permitted the escape of a thin, clear fluid, and, thereafter, the drum appeared to be perfectly normal.

HISTORICAL REVIEW.

Buck[1] described six instances of cysts of the tympanic membrane in his textbook published in 1889. Of these, one was described as being between the substantia propria and the outer dermoid layer in the very center of the drum membrane and measuring nearly 3 mm. in diameter. The cyst contained "apparently pure serum, unmixed with blood" and disappeared soon after the contents had been evacuated. No further record of this type of cyst (or bleb) appeared for over 60 years. Politzer's[2] comprehensive monograph on the

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membrana tympani did not include them. Other varieties of cysts were briefly discussed in the textbooks written by Heiman[3] and Keeler.[4] Fowler[5] mentioned bleb-like structures of the drum in 1947, but made no reference to blebs at the umbo.

Fig. 1. Artist's drawing of the right tympanic membrane showing a dermal bleb at umbo.

In 1952, Wible[6] directed attention to a "frequently observed change seen at the umbo in cases of secretory otitis media" and reported his observations in three patients. He described a fluid collection at the umbo which sometimes exhibited a fluid level. He stated that some of the collections of fluid were subdermal and others submucosal. His patients complained

of difficulty in hearing and egophony and were relieved by incision and aspiration of the fluid.

Friedman[7] published his findings in five patients with "hydrops at the umbo," in 1954. The blebs were incidental findings in two patients, while the others were thought to have caused pain, tinnitus, and stuffiness of the ear. He theorized that the fluid could be a transudate or an accumulation of lymph.

Epidermal and mucosal blebs at the umbo have also been observed by Lathrop[8] and Arnold.[9] Because the blebs in their patients were asymptomatic without exception, they regarded them as incidental findings. Their cases have not been recorded in the literature.

Although vesicular lesions at the umbo have been reported,[1,6,7] the pathology apparently has not been investigated. No record of a histopathologic study or even an examination of the fluid has appeared in the literature. Wible[6] discussed two separate types of lesions, "subdermal" and "submucosal," as though they were one entity. It is my belief that he actually observed two distinctly different conditions, each having its own significance. Umbo blebs occur on the epidermal and mucosal surfaces of the tympanic membrane, but there is no evidence that they have the same etiology or pathology.

DESCRIPTION OF UMBO BLEBS.

These benign, asymptomatic blebs are invariably situated in the center of the drum. They vary in size from 1.5 to 4 mm. in diameter and appear as a smooth elevation, often glistening, and brighter than the rest of the drum (see Fig. 2A). A bleb may resemble a droplet of dew on a gray petal (see Fig. 2B). The margins are frequently marked by a faint ring of pallor. These blebs are common, yet easily overlooked. They are probably dismissed by many otologists as an unusual light reflex or an optical illusion!

Of the 66 umbo blebs observed in the past five years, 63 were external to the manubrium of the malleus, and only three were medial to the manubrium. The mucosal blebs are much more difficult to recognize, and some may have escaped

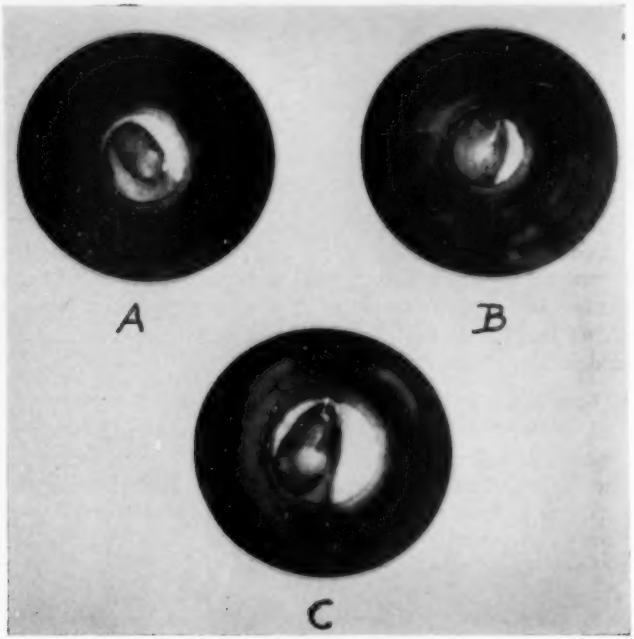


Fig. 2. External umbo blebs.
A. Highlights give bleb a glistening appearance.
B. Very delicate bleb. Note incomplete ring of pallor.
C. Bleb undergoing absorption. Note wrinkled margins.

detection.* Both types of blebs tend to disappear spontaneously in a matter of several weeks or months. As they diminish in size, the margins become wrinkled (see Fig. 2C), Recurrences have not be observed. There was nothing to suggest an intra-vesicular fluid level in any of my cases.

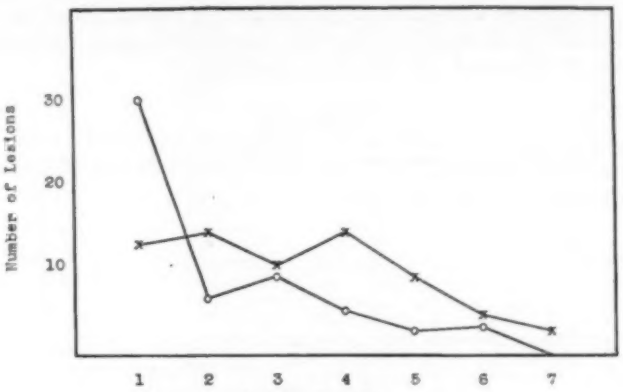
INCIDENCE.

Sixty-six umbo blebs were found in 58 patients during 24,000 examinations, giving a relative incidence of one for

* The number of mucosal blebs in this series is so small that the study has not been extended to include them other than to record their existence. It is assumed that they are a separate entity, having a different etiology and pathology from the epidermal blebs.

every 364 examinations.* A tabulation of the distribution according to sex revealed that 42 of the patients were female and only 16 were male. The age incidence was fairly uniform up to the age of 50 (see Chart I). This is in contrast with the acute vesicular lesions which are much more common in the first decade of life. The seasonal incidence of umbo blebs is indicated in Chart II.

CHART I



Umbo Blebs X Myringitis Bullosa O

AGE INCIDENCE

During the same period, in 52 patients with myringitis bullosa (both serosa and hemorrhagica) were seen. The age and seasonal incidence of these cases is also indicated in Charts I and II, respectively, to illustrate the contrast between myringitis bullosa and umbo blebs. Admittedly, the number of cases may be too small to give an accurate concept of seasonal, age, or sex incidence.

ETIOLOGY.

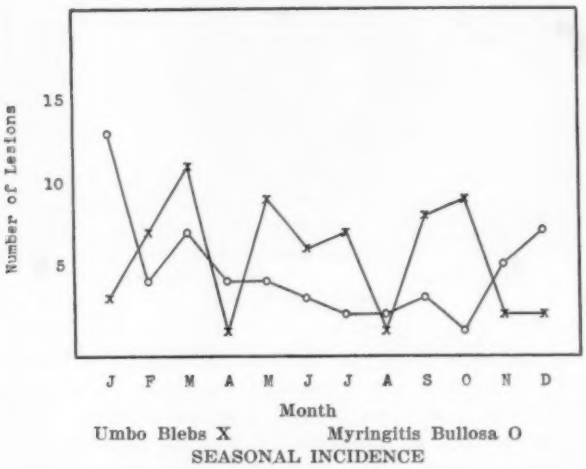
The etiology remains unknown. It is interesting to note, however, that the three periods in which most of the blebs

* The relative incidence was computed by dividing the total number of patient visits (24,000) by the total number of blebs (66). Although each bleb was counted only once, many patients, both with and without blebs, made repeated visits during the five-year period represented, and each visit was recorded. The true incidence is, therefore, considerably higher.

were observed also correspond to the time of high concentration of tree, grass, and ragweed pollen (see Chart II). Of the 58 patients having blebs at the umbo, 12 had confirmed allergic manifestations. Nevertheless, the lack of ear symptoms and the character of the fluid in the blebs (see Pathology) argue against allergy as an etiologic factor.

Neither trauma nor "local noxious stimuli"[7] appeared to be responsible for bleb formation. Circulatory disturbances were not considered to be factors in producing blebs. There is an abundant blood supply along the manubrium, and other evidence of impaired circulation is lacking.

CHART II



Umbo Blebs X Myringitis Bullosa O

SEASONAL INCIDENCE

There has been nothing in any of these cases to suggest a diagnosis of herpes. It has been stated,[5] without supporting detailed evidence, that blebs of the drum are occasionally due to tuberculosis. This is open to question, since tuberculosis is not characterized by bleb formation. There was no evidence of tuberculosis in any of my patients.

There was no apparent relationship to acute upper respiratory infections. This is in contrast with the fact that, in this

series, 39 out of the 52 patients (75 per cent) with myringitis bullosa had associated acute upper respiratory infections. Unlike Wible's[6] patients, only one patient in this series had secretory otitis media at the time the bleb was under observation.

In considering the etiology of this lesion, the fact that epithelial growth progresses from the umbo toward the periphery assumes significance. The position of the lesion suggests that its formation may be related to this pattern of regeneration, peculiar to the tympanic membrane.

PATHOLOGY.

These blebs are exceedingly thin-walled. It was only after many unsuccessful attempts that a specimen was obtained and sectioned (see Figs. 3-4). The pathologist's description follows:

"Microscopic: The specimen represents a longitudinal section perpendicular to the surface of the drum. Only the outer epithelial covering is seen. It is composed of several layers of epithelial elements, varying in shape from cuboidal to flat. They produce a bright pink layer of keratin-like material in which occasional flat, elongated, pyknotic nuclei can be seen and which are covered on one surface by delicate, pinkish-stained keratin lamellae. The substantia propria and the mucous membrane are not contained in the specimen.

"A long intra-epithelial slit is present in the central portion. The base of the slit is a linear membrane composed of a few layers of compressed stratified squamous cells. The roof, however, is undulating and consists of the continuous upper layer of epidermis covered by keratin lamellae. On either side, the upper and lower layer of epithelium can be seen to merge into the intact epithelial covering of the drum. Although the vesicle is completely empty in all sections, it cannot be regarded as an artefact; for, as indicated by its undulation, the continuous epithelial layer of the roof is much longer than the corresponding basal portion. This configuration is indicative of a collapsed pre-existing vesicle. There is no evidence of intercellular edema or inflammation in the specimen examined."

The fluid within the blebs was crystal-clear except for two instances in which it was burnt orange in color. No cellular elements or organisms were demonstrated in any of the smears. The fluid contained large, occasionally confluent, drops of a sudanophilic material. Smaller, irregular particles polarized light, but the Liebermann-Buchardt reaction for cholesterol was negative. No crystals were observed in any of the smears. The thin, oily fluid will not dry, even after standing for several days.

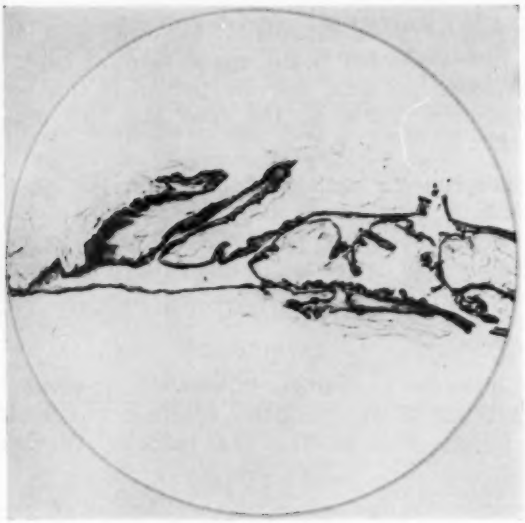


Fig. 3.

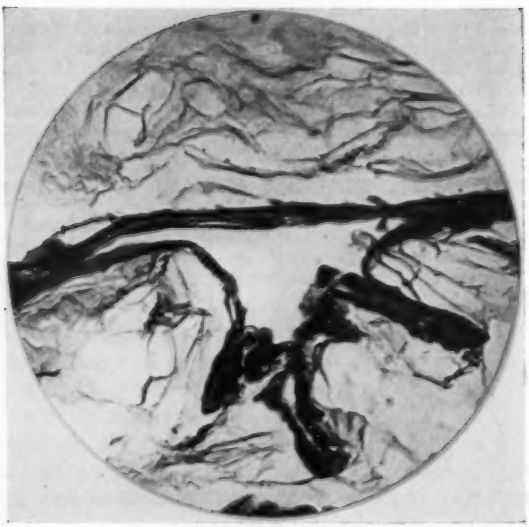


Fig. 4.

It is evident from the above that previous writers[1,6,7] have erroneously assumed that umbo blebs are necessarily subdermal (or submucosal). The section illustrated in Fig. 3 clearly shows the lesion to be intra-epidermal. The lack or scarcity of circular fibers[7] in the substantia propria at the umbo cannot be a limiting or contributing factor, because the substantia propria is not involved.

Friedman[7] referred to this condition as "hydrops at the umbo." Hydrops is taken directly from the Greek word *hydrōps*, meaning dropsy or an excessive accumulation of clear, watery fluid. Since the fluid in the blebs is oily, it is evident that the term "hydrops at the umbo" is inaccurate and should not be employed.

Wible[6] has reported a fluid level within the blebs in some of his patients. It is difficult to explain the presence of air or other gases in a non-inflammatory vesicle. In view of the above findings, it seems more probable that he observed a line separating layers of watery and oily fluid. The difference in the refractive index of water and lipoid might give the illusion of a line of demarcation between fluid and air.

External blebs at the umbo probably represent a primary degenerative lesion of the epithelium. No inflammatory exudate was observed. Although actual necrosis of epithelial cells was not demonstrated and the remaining epithelial layer was coherent, degeneration is presumed. The fluid contained lipoid that cannot be accounted for except by disintegration of epithelial cells. Eggston and Wolfe[10] have attributed bleb formation to hydropic degeneration or edema. This alone does not account for the formation of lipoid blebs. Since lipoid-containing, intra-epidermal blebs are not discussed in the textbooks on histopathology of skin,[11,12] they are apparently unique to the umbo. Primary degeneration is merely inferred since the etiology remains obscure. Additional investigation may provide more definitive data.

SYMPTOMS.

Significant symptoms are strikingly absent. When there is a complaint referable to such an ear, it can almost be

assumed that the bleb is incidental. This observation has been borne out by Lathrop[5] and Arnold.[6] Four patients in this series had co-existing deafness. Two had clinical otosclerosis, bilaterally, and had been under observation by one of my associates for years. Each of the other two patients had a long history of perceptive deafness. Not one of the patients noted any change in hearing acuity in relationship to the blebs. One patient complained of vertigo. Incision into the bleb did not alter the vertigo.

A few patients complained of pain in the ear. In two of these, the blebs were bilateral, but the pain unilateral. In another, the pain was of dental origin and did not subside after the bleb was evacuated but disappeared after the extraction of a carious molar. In no instance was there any sign of otitis media or myringitis. The benign nature of these blebs is indicated by the fact that they contain lipoid; show no evidence of inflammation, even microscopically; and often persist for several weeks.

It is conceded that a large mucosal bleb could cause a conductive loss of hearing. Only 2 mm. separate the tip of the manubrium from the promontory, and a bleb that exceeded 2 mm. in diameter might produce the sensation of stuffiness and egophony as described by Wible.[6] This relationship should be remembered, since a mucosal bleb could explain an otherwise obscure conductive deafness, and the symptoms could easily be relieved by incision into the bleb. A large epidermal bleb, by virtue of its mass, might be expected to produce symptoms, but neither type of bleb has been responsible for symptoms in any of my patients.*

DIFFERENTIAL DIAGNOSIS.

The diagnosis of an umbo bleb should present no problem. They are common and, once recognized, will be noted with increasing frequency. External (epidermal) blebs should be easily differentiated from internal (mucosal) blebs. The only

* Since this manuscript was prepared, a patient with a large mucosal bleb has been seen. Her complaints, as hypothesized above, were those of stuffiness in the ear and egophony. Relief was obtained promptly after the bleb was incised.

conditions with which they may be confused are cysts and myringitis bullosa, both of which are rarely seen at the umbo. The fluid within external umbo blebs is oily rather than serous or hemorrhagic; further, no symptoms have been observed that could be definitely related to the formation of lipoid blebs at the umbo.

TREATMENT.

It is questionable whether or not any treatment is necessary. At most, simple incision into the bleb is all that is indicated, and this should be reserved for patients with definite complaints. Incision can be expected to eliminate the bleb but not necessarily pain, tinnitus, vertigo, or deafness. Friedman[7] was unable to exhibit a significant change in hearing after he dispersed the fluid by the application of pressure with a cotton-tipped applicator.

CONCLUSIONS.

Non-inflammatory umbo blebs containing lipoid material were the most common vesicular lesions of the tympanic membrane in this series. Since they are asymptomatic, the finding was usually incidental to doing a complete examination. The lack of symptoms probably accounts for the fact that most otologists are unfamiliar with their existence. The significance of these blebs at the umbo remains unknown at this time.

SUMMARY.

Blebs at the umbo were found in 66 ears in 24,000 examinations, an incidence of 1:364. The external blebs are smooth, rounded, glistening intra-epidermal collections of clear, oily fluid and were observed much more frequently than mucosal blebs in this series. Definite symptoms were significantly absent in all cases. It is suggested that these noninflammatory blebs represent a localized degenerative process resulting in the dissolution of epithelial cells. The etiology remains to be determined.

Acknowledgment: My sincere appreciation is expressed to Dr. Paul Kimmelstiel for his description of the pathology and for assistance in determining the nature of the vesicular fluid.

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STUDIES ON PER-STIMULATORY ADAPTATION IN VARIOUS GROUPS OF DEAFNESS.*

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INTRODUCTION.

Studies in auditory fatigue have hitherto been concerned mainly with the determination of the amount of threshold shift measured shortly after cessation of a stimulus. The magnitude of loss in loudness level experienced during a continuous stimulus has only recently aroused attention in experimental literature. This loudness change was termed by Hood[6] per-stimulatory fatigue or adaptation, both terms being used interchangeably.

Hood's experiments, so lucidly described, brought out one important clinical implication. It seemed that most of the patients suffering from Menière's disease with complete recruitment experienced an abnormally large loudness level loss during three minutes' stimulation. This finding suggests that the measurement of per-stimulatory adaptation might have some use as a method of classifying hearing impairments into clinical subgroups, in about the same fashion as the recruitment tests proper are presently used.

It has been reported earlier that the recruitment phenomenon is accompanied with a poor articulation score. The loss in speech discrimination, however, depends considerably on the specific test items and the language used. Hirsh, Palva and Goodman[7] demonstrated with American PB words that a hearing loss for speech of more than 20 db in recruiting ears parallels a poor discrimination ability. In Finnish test material[11] speech function was not seriously impaired until a 40 db hearing loss at threshold was exceeded. It also appeared that a slight hearing loss of less than 25 db did not affect the discrimination ability.

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From a study[7] dealing with difference limina to intensity it became apparent that difference limina, measured by a two-tone comparison, did not differ significantly in the various subgroups of deafness, although in speech function there were no doubt large differences between these groups.

We pointed out that, for good speech intelligibility, many factors probably are important: Ability to detect rapid changes in intensity, in fundamental frequency, and in spectrum. One would presume also that the ear would neither fatigue so easily as to render speech sounds too weak in loudness nor become temporarily fatigued by the louder speech elements rendering the soft formants inaudible.

In an earlier paper[13] I tried to imitate the possibly fatiguing effect of loud vowels upon weaker consonants, by introducing into the ear an interrupted white noise and in the presence of this noise measuring the thresholds for pure tones. It is quite clear that the audibility of pure tones and the intelligibility of speech are things apart.

It was intended primarily, however, to ascertain whether the interrupted noise would show the effect of per-stimulatory fatigue upon the tones. At the highest available interruption, 12 per second, this was not the case. When speech and noise were introduced simultaneously into the same ear, the perceptively deaf ears were usually severely affected, but the conductively deaf ears were not affected. There was no clear difference between the recruiting and non-recruiting perceptive groups, the speech score being frequently lowered in both.

It may be noted here that a differentiation between the perceptive subgroups on the basis of speech tests alone cannot always be successfully done. Although the non-recruiting ears usually show moderately good speech discrimination, there are always ears showing poor intelligibility. In connection with acoustic tumors Schuknecht and Woellner's[16] hypothesis was, actually, that a certain minimum number of functioning nerve fibers is one of the essential requirements for good speech function. It seems evident that in seeking to explain a poor articulation score, one is dealing with so many different factors that one explanation will not suffice.

This study has been carried out in an attempt to determine the changes in loudness level during a continuous stimulus in various groups of deafness and to relate these changes to the recruitment phenomenon. Secondly, the changes in adaptation are compared with the data of speech intelligibility to learn whether or not these two phenomena can be correlated.

BRIEF REVIEW OF EARLIER STUDIES.

One of the first to study the development of a per-stimulatory adaptation was de Maré.[10] He obtained a loudness balance between two pairs of 475 cps tones of 0.4 seconds duration alternately in the ears. These tones followed one another after an interval of 2.7 seconds indefinitely. When the free interval in the test ear was substituted for a 500 cps tone of the same 88 db intensity, and another balance with the control ear was obtained, there was a loudness level loss of about 30-40 db.

If the fatiguing tone of 500 cps was followed by a pause of 0.7 seconds before the balancing pair of tones appeared, then a loss of only about 5 db in the loudness level was observed. It was pointed out by Hood[8] that the first procedure is essentially comparable to his own per-stimulatory adaptation experiments, as the two frequencies mentioned were so close to each other. In the second experiment the silent period of 0.7 seconds was sufficient to allow the normal on-effect to appear and result in about equal loudness level between the ears.

Hood's monograph[8] on auditory fatigue describes the first detailed and well-controlled experiments on per-stimulatory adaptation placing it in a distinct relationship with neurophysiological phenomena in general. Hood's technique in normals was as follows: A continuous tone was given to the test ear and, from time to time, a balancing tone of 10 seconds' duration was led to the other ear.

The balance, as judged by the observer, was automatically registered on a graph. The measurements were made with tones of 500, 1000 and 2000 cps at 20 to 100 db sensation levels. Hood concluded that per-stimulatory fatigue develops at

a fairly constant rate and quite slowly, reaching a maximum in about three minutes, and increasing with the sensation level. The tests were carried out, using four normally hearing subjects. It appeared that the amount of per-stimulatory adaptation was individually different, but generally about 30 db at 80 db sensation level (see Figs. 3-5).

In hard-of-hearing subjects, and also in some normal ears, another procedure was used. The subject was given a continuous tone of 70 db above normal threshold in both ears, and he was instructed to control the attenuator of one (better) ear to maintain a loudness balance between the ears.

Hood mentioned that in 25 cases of Méñière's disease, 20 showed abnormal fatigability in the diseased ear, which manifested as a decrease in the level of the balancing tone by 10 to 50 db. In conductively deaf persons, and in normals, the balance tracing pursued a steady course without fatigue during the three minutes of the test.

The two different techniques of measuring per-stimulatory adaptation bear an important implication. If a fatiguing tone is responsible for about 30 db loudness level loss in the normal ear when the comparison is made with an *interrupted* balancing tone, then when both the fatiguing and the balancing tone are *continuous*, the loudness level loss will still be present without anything present to indicate this on the self-recording audiogram.

If, in a recruiting ear, this latter type of adaptation test gives a reduction of say 40 db, we should be able to measure a considerably larger loss in loudness level with an interrupted balancing tone. The validity of this implication was not tested by Hood.

Hood's experiments suggest that after the first three minutes' stimulation there is no noticeable change in the loudness sensation, but a discharge at a steady rate will follow; this finding is also supported by the action potential measurements of Derbyshire and Davis[1] in the auditory nerve of a cat. Van Dishoeck's citation of van Gool's work,[2] on the other hand, shows that the loudness adaptation may not be complete until after six to eight minutes' stimulation.

TESTING PROCEDURE AND APPARATUS.

Air and bone conduction audiogram were measured in the usual manner. In these tests both ears were covered with air conduction receivers, and masking with a thermal noise was employed according to the principles described earlier.[12] The source for pure tones was a Peters SPD 2 audiometer with PDR 10 earphones, which was calibrated in terms of the sound presure level at 60 db reading and relative to a fixed input of 0.1 volts at various frequencies. The values relative to the zero level of the audiometer are presented in Table I.

TABLE I.

SOUND PRESSURE LEVELS OF THE PETERS SPD-2 AUDIOMETER AT 0-DB HEARING LOSS (DB ABOVE 0.0002 MICROBAR).

Frequency	125	250	500	1000	2000	4000	8000
	55	43	25	17	15	15	18

Recruitment testing was done by applying tones of 1.5 seconds' duration with an interval of 0.5 seconds alternately to the ears according to the principles of Fowler's balance test. If this were not possible Reger's monaural test was used.

Speech reception thresholds were measured with the Finnish Test List No. III, containing easier words of trochee type, recorded one by one, by decreasing the intensity manually 5 db after five successive words, until the subject did not hear a single word correctly. Speech discrimination was measured with Test Lists I or II containing more difficult trochee type words, using 30 words for each ear. The intensity for discrimination was kept, as far as possible, 40 db above the SRT.[11]

At the beginning of these studies the apparatus for measuring per-stimulatory adaptation was quite simple. The two earphones in a headband were fed separately by two audiometers, a Peters SD-2 and an Amplivox M-60. The differences in calibration between these two and the testing audiometer Peters SPD-2 were taken into account.

A continuous tone was given into the test ear and a balance was obtained for manually interrupted test tones, about two seconds long, in the balancing ear. This balance was obtained right at the beginning of a three minutes' fatigue period and then after one, two and three minutes' stimulation.

For about one-third of the hard-of-hearing subjects a Békésy type self-recording audiometer was available. This audiometer has two separate channels in ordinary use: One for the tones and another for the masking noise. In the first channel the tone is controlled automatically in 1 db steps over an intensity range of 140 db at a rate of 2.3 db per second as the subject presses a button. In the second channel the stimulus can be controlled manually by the operator in 5 db steps. These two channels can also be fed by a separate sound source, and the intensity controlled before the attenuators with a Pegel voltmeter.

For testing per-stimulatory adaptation, a Peters SPD-2 audiometer was used to feed both channels simultaneously with a pure tone that could be controlled in both earphones as described above. A block diagram of the apparatus is shown in Fig. 1.

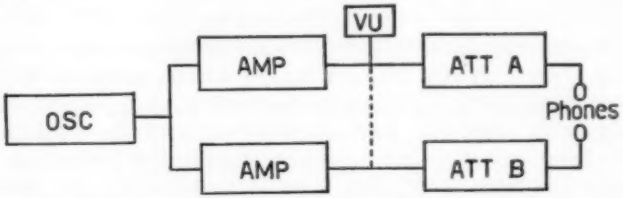

Fig. 1. Block diagram to illustrate the arrangement of equipment for measuring per-stimulatory adaptation. The tone intensity is controlled in both channels with a VU-meter before the attenuators. The fatiguing tone is led through attenuator B, where it is controlled manually in 5 db steps by the operator; the balancing tone is led through attenuator "A," where it is controlled automatically in 1 db steps by the subject.

The technique described for the simpler apparatus was used, the sole difference being that the balancing tone was kept *continuous* for 15 seconds, and the writing apparatus recorded its excursions automatically during this loudness match. In a few cases the tones in both ears were kept con-

tinuous during the entire testing period, and a continuous tracing from the balancing ear was obtained.

DIFFICULTIES IN THE DETERMINATION OF LOUDNESS ADAPTATION.

A proper loudness balance test is made by comparing two tones of equal duration presented alternately at a short interval to both ears. On the other hand, many audiometers are equipped with a loudness balance control where the same tone is delivered simultaneously to both ears, and an attenuator allows a reduction in the intensity of the tone in one earphone. A position for the attenuator that the subject feels corresponds to a sensation of equality between the ears is sought.

The difficulties in this type of loudness balance test were pointed out successfully by Hirsh.[6] When a tone is simultaneously heard in both earphones, the loudness comparison is rendered uncertain because the tones have an unitary localization, usually at the center of the head. In a sense, one is substituting a judgment of central localization for a loudness balance judgment. There will probably be some differences between the various frequencies; the judgment ought to be easier in the high tones because the differences in intensity are more important for localization at these frequencies, whereas for low tones the difference in the time of arrival (or phase) is more important.[16]

The measurement of per-stimulatory adaptation must be made in the same fashion. A continuous tone is introduced into the test ear and either manually or automatically with a recording apparatus, a balancing tone is brought for a short time into the control ear. The fatiguing tone in the test ear cannot be discontinued without seriously interrupting the experiment, and, therefore, the test loses in accuracy as compared with binaural alternate balance.

A preliminary experiment, therefore, was performed on normally hearing persons to test the magnitude of the variations in intensity for tones judged equal in loudness. A tone of

60 and 80 db sensation level was given to both ears at 4000, 2000, 1000 and 500 cps in the order mentioned, to avoid overlapping fatigue,[17] and the subject was instructed to maintain equal loudness in the ears.

The recording of the variability of the tone intensity in the balancing ear was made automatically with a Békésy-type audiometer during a one-minute test period at each frequency. The width of the excursions did not differ appreciably at the four frequencies. In a few instances it was about 15 db, but generally it seemed that a width of 10 db was a fair average (see Fig. 2).

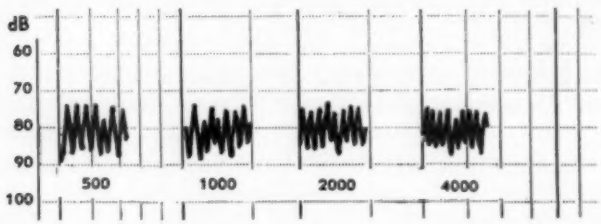

Fig. 2. Simultaneous bilateral loudness balance test during a continuous stimulation of 1 minute at 80 db sensation level. The width of the excursions expresses the intensity range that is needed to make the balancing tone in one ear to sound alternately softer and louder than the 80 db steady tone in the other ear.

Subjectively, however, judgment was mostly felt to be easiest at 4000 cps where the tones could be heard separately in the ears. The more the frequency was lowered, the more central was the localization, and the tone seemed to shift, not in the ears but in or above the head.

For an average listener, therefore the accuracy of a simultaneous bilateral loudness balance test seems to be around ± 5 db and definitely less than ± 10 db. When this is applied to a test for per-stimulatory adaptation, there should be an equal distribution of the values around the initial balance tracing, providing no adaptation occurs. In the case of a loudness loss due to adaptation, the subsequent tracings should be lower than the initial balance value. A loss of 10 db or

more would be indicative of a real loudness change and not due to an experimental error.

PER-STIMULATORY ADAPTATION IN NORMAL EARS.

At the beginning of this work Hood's[8] figures for the amount of auditory adaptation were taken as a reference, and then proceeded dir ctly to testing hard-of-hearing patients. It soon appeared, however, that the amount of adaptation obtained in deaf subjects differed so much from Hood's figures that a validation work on normal material seemed also necessary. This was done entirely with the Békésy audiometer. The subject himself controlled the intensity in the balancing ear, and the recording was made automatically.

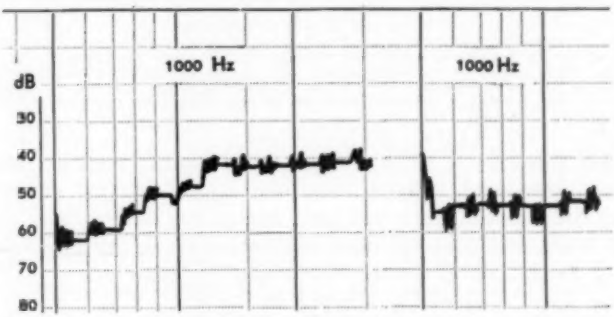

Fig. 3. Two different types of behavior in per-stimulatory adaptation test at 80 db sensation level. In the left, adaptation amounts to 25 db; during the first 5 minutes, remaining thereafter unchanged for another 5 minutes. In the right there is practically no adaptation during the 7 minute test. Each tracing is made during a 15 second balancing period and the interval between succeeding tracings is 1 minute.

All test persons had some experience in threshold testing, and their air conduction curves measured on different days showed little variation. Two sensation levels, 60 and 80 db (see Figs. 3, 4) were used. The subject was first given a tone at 60 db level to both ears, and he was requested to maintain a balance between the ears. After 15 seconds the tone in the balancing ear was discontinued, and the tone in the test ear was kept continuous for some minutes. After each minute a 15 seconds balancing period was introduced, which re-

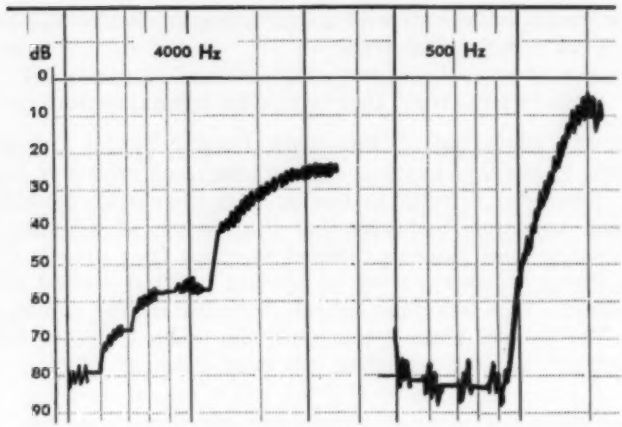

Fig. 4. Two different types of behavior in per-stimulatory adaptation test at 80 db sensation level. In the left, adaptation amounts to 25 db; in the right there is no adaptation loss at all during 3 minute testing period. The post-stimulatory threshold tracing does not reach normal level during 2 minute recovery period; at the right the recovery to normal values is rapid.

corded aumatically the development of per-stimulatory adaptation. For each subject, only one sensation level at 4000, 2000, 1000 and 500 cps was used during one day to make sure that there was no remaining adaptation from previous tests.

TABLE II.

ADAPTATION LOSS IN NORMAL EARS AFTER CONTINUOUS STIMULATION OF 4 MINUTES AT 60 DB, AND 3 MINUTES AT 80 DB SENSATION LEVEL.

Frequency	Sensation Level	Average Intensity Level	Mean of Adaptation Loss	Standard Deviation	Number of Subjects
500	60	78	6.4	9.7	9
	80	98	4.8	8.4	12
1000	60	74	8.6	6.1	15
	80	94	2.2	6.4	12
2000	60	73	4.7	5.3	9
	80	93	4.8	7.5	12
4000	60	75	5.5	6.2	9
	80	95	3.9	9.9	12

The results for normal subjects are presented in Table II and graphically in Fig. 5. It is apparent: first, that the

individual results varied within rather wide limits. Some showed considerable adaptation of the order of 20 db; in others there was no loudness loss at all and the balancing tone showed variations of the order of ± 5 db around the value of initial balance. This is quite in accordance with the assumption made earlier for cases, in which no per-stimulatory loudness loss develops. Second, it can be seen that the figures for the two different sensation levels are essentially the same, *viz.* the loudness level loss does not increase with increasing intensity.

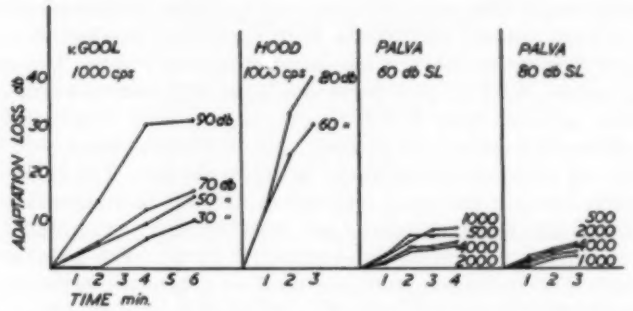

Fig. 5. Adaptation loss as a function of sensation level; V. Gool's figures are drawn from van Dishoeck's[2] Fig. 3, where the term "loudness in db" is interpreted as to mean sensation level. Hood's[3] curves are reproduced from Fig. 15. There is a substantial difference between Hood's and the two other studies at lower sensation levels during a 3 minute stimulation.

It must be pointed out that these figures cannot be considered sufficient for a detailed study of per-stimulatory adaptation in normal ears. For that the material is too limited, and the range of different sensation levels is not adequately covered; but for the purpose of this paper they are representative of normal values as compared with values for hard-of-hearing ears at the same levels.

Large individual variations in per-stimulatory adaptation were also observed by Hood, who reported a difference of 26 db between two subjects at 100 db sensation level. In two of the four ears studied by him, the loss in the loudness level at 80 db sensation level was between 20 and 30 db for 500-2000 cps tones. It is probable that a larger sample of cases

would also have disclosed subjects with a minimal loudness change.

The standard deviation in each group (see Table II) shows quite large values, which suggest that a loudness level loss of the order of 30 db (more than 3 SD's) is indicative of a really abnormal sensitivity to continued stimulation.

PER-STIMULATORY ADAPTATION IN DEAF SUBJECTS.

The patients studied for per-stimulatory adaptation were divided into clinical groups on the basis of the criteria commonly used. The conductive group includes unilateral cases of chronic middle ear disease with good bone conduction and without recruitment, and also some otosclerotic ears. The second group, that of perceptive deafness with recruitment, includes patients with Méñière's disease, cochlear degeneration or acoustic trauma. All of them showed lowered bone conduction and complete recruitment in Fowler's test. The cases of equally low air and bone conduction without recruitment and without any signs of middle ear involvement[14] were included in the third, perceptive non-recruiting group. The results of speech discrimination were not used as diagnostic criteria in this study.

The conductive group comprised 18 ears. In these cases a fatiguing intensity of 80 or 100 db above normal threshold was used to stimulate the impaired ear for three minutes. The sensation level, however, could not always be raised to the 80 db level because the conductive component reduced the intensity in the inner ear.

In the recruiting perceptive group 51 ears were tested, mostly at a sensation level of 80 db above normal threshold. In more severe cases a 100 db level was also used (four ears). Because of the positive recruitment phenomenon the loudness level in these ears should be comparable to normal or nearly normal cases.

The perceptive non-recruiting group consists of only nine cases, mostly tested at a sensation level of 80 db, sometimes 100 db above normal threshold. The loudness level in these ears should be comparable to those in the conductive group.

The loudness level loss due to per-stimulatory adaptation is presented summarized in Table III. One can see that the loss in the conductive and non-recruiting perceptive group is of the same magnitude, and does not differ appreciably from the normal values in Table II. In the perceptive recruiting group, however, there are some ears, in which the loudness loss due to adaptation is definitely larger than could be expected, although the change in many cases is comparable to that of normal.

TABLE III.

MAGNITUDE OF ADAPTATION LOSS AND ITS OCCURRENCE IN VARIOUS GROUPS OF DEAFNESS.

Type of Hearing Loss	Adaptation Loss in db											Total Number of Cases
	-5	0	5	10	15	20	25	30	35	40	45	
Conductive Group		5	4	6	2	1						18
Recruiting Nerve Group	1	11	11	13	5	2	1	3	1	1	2	51
Non-recruiting Nerve Group		1	3	4		1						9

It was pointed out earlier that in Finnish speech tests a lowered discrimination score is usually associated with cases of recruiting perceptive deafness showing a threshold loss of 40 db or more. Table IV is presented to illustrate the relation between low discrimination score and adaptation loudness loss. It includes all cases of poor speech intelligibility with detailed audiograms between 500 to 4000 cps. It can be seen that among these 11 cases there are four with 30 db or more loss in loudness level; in the remaining seven cases stimulation for three minutes did not cause abnormal change in the loudness levels.

The three cases of Table III that show 30 db or more loss in the loudness level include two, in which the threshold loss was less than 40 db. In one case with a 35 db loudness level loss at 4000 cps, the threshold was 50 db below normal, but the other frequencies showed a loss only around 20 db in the speech range. Speech intelligibility was normal in all these cases.

TABLE IV.

CASES WITH LOWERED SPEECH DISCRIMINATION RELATED TO ADAPTATION LOSS.

Case	Threshold Audiogram				Adaptation Loss				Speech Discrim. Per Cent	IL of the Fatiguing Tone (re 0-level)	Diagnosis
	500	1000	2000	4000	500	1000	2000	4000			
SA	60	70	55	55			0		3	80	Ménière
OP	65	65	50	45	0	5	5	5	3	80	"
LK	25	75	65	60	15	5	10	15	0	90, 80	"
OH	40	40	40	20		15			61	80	Ac. trauma
SV	50	55	40	40	10	10			60	80	Ménière
MH	60	70	65	55		30			30	90	"
LH	45	55	50	65	10				68	80	Cochlear deg.
MM	55	65	55	45			45		70	80	Ménière
AM	45	50	50	30		40	10		74	80	"
LL	70	70	55	75			30		68	80	"
MA	60	60	30	25		20			22	90	"

In the conductive and in the non-recruiting perceptive groups speech intelligibility was normal in all cases.

Two cases only were tested with a continuous tone in both ears during the whole testing period. These cases showed about 5 db loudness adaptation in the previous test with interrupted test tone in the balancing ear. The tracing obtained during continuous stimulation in these recruiting ears showed essentially the same result, although the patients complained that judgment was more difficult.

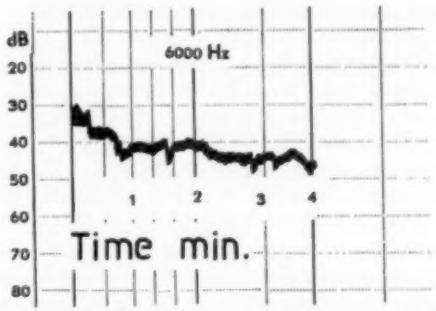

Fig. 6. A continuous threshold tracing at 6000 cps during 4 minute testing period. The threshold is lowered by about 10 db and the width of the excursions remains the same, or is somewhat reduced.

In four cases a continuous threshold tracing was recorded during a four minute period at a frequency showing positive recruitment. The tracing had an even course in one ear, one showed deterioration of threshold by 5 db and two a further loss of 10 db (see Fig. 6). No increase in the width of the excursions was noted. In the case with 10 db loss, on the contrary, the threshold variability decreased clearly. This is in opposition to the view held by Dix and Hood.[3] Studies of this question are to be continued with a greater number of subjects.

DISCUSSION.

The figures for per-stimulatory adaptation in normal subjects suggest that the development of adaptation is not related to the intensity level, as far as the two sensation levels of 60 and 80 db are concerned. This finding is in accordance

with van Gool's[4] results, but differs from the opinion of Hood, who regards the increasing intensity level as an important parameter for an increased per-stimulatory adaptation.

Hood feels that, contrary to post-stimulatory fatigue, there is no critical intensity for an abnormally large increase in per-stimulatory adaptation. Van Gool's results, on the other hand, suggest that there is a traumatizing intensity after which the per-stimulatory adaptation increases disproportionately to previous levels. This level probably is higher than the 90 db sensation level, although no precise intensity scale can be found in the text cited by van Dishoeck.

At 80 db sensation level a 10 db adaptation loss would mean a loudness loss from 25 to 12 sones, and a 20 db decrease correspondingly to 6 sones. This would already be a two- or fourfold decrease in loudness. One would suspect that normal adaptation could hardly be responsible for more loss than this.

It was pointed out earlier that, according to Hood's results, there should be quite large adaptation changes. He considers that a loss of about 30 db is obtained in normal ears with an interrupted balancing tone at 80 db sensation level. In hard-of-hearing cases a loss in loudness level of 10 to 50 db was demonstrated with a continuous balancing tone.

This latter technique does not take into acount the adaptation which, logically, is bound to occur in the continuously stimulated balancing ear. Providing the losses in the loudness levels are simply additive, adaptation values between 40 and 80 db ought to be obtained with an interrupted balancing tone, because no interfering adaptation would develop in the balancing ear. This amount of loss would mean that in many cases the tone would almost vanish from the tested ear during a three minute fatiguing period.

The technique with an interrupted balancing tone in the good ear did not reveal adaptation losses greater than 45 db in this series. On the basis of the standard deviation in Table III, one is inclined to consider adaptation losses of 30 db or more abnormally large. These occurred only in the group

of recruiting perceptive deafness, totalling 7 or 14 per cent. In only one case did the patient feel that the test tone nearly vanished from her ear during the three minute fatiguing period.

The findings reported by Hood,[8] Hallpike and Hood,[5] and Dix and Hood[3] are confirmed in a way. The results differ considerably, however, in the magnitude of the adaptation as well as in the percentage of recruiting subjects in whom this phenomenon was found. It seems that the role of the loudness level loss has hitherto been overestimated, and that conclusions have been drawn somewhat prematurely; however, when there is an abnormally large loss in loudness level it seems to be associated with recruiting perceptive deafness.

Speech discrimination and abnormally large loudness adaptation do not seem to be related. Poor speech intelligibility can be found in cases of perfectly normal adaptation loss. This is understandable when we think of the very short time needed for one word to be "on." Although the patients do not repeat the words correctly, they often spontaneously remark that they are quite loud; furthermore, the Finnish records do not contain any introductory phrases, but the words follow each other at intervals of five seconds. The recovery period for short time fatigue should, therefore, also be sufficiently long.

It seems that the decrease in loudness for speech sounds does not by any means explain poor intelligibility. Nor is the temporary fatiguing effect of loud vowels upon weaker consonants supported by a comparison between Finnish and American test material. In Finnish words, the role of the vowels is clearly more important than in the PB words; yet the former are more easily understood.

Licklider and Pollack's studies[9] with clipped speech waves, even when reduced to parallel straight lines, resulted in a good articulation score. This has cast considerable doubt on the theories of disturbed loudness values at various frequencies as the sole cause of poor speech intelligibility; however, if the clipper was preceded by a low-pass filter (integrator), suppressing the spectrum 6 db per octave, a large discrimina-

tion loss followed. This situation might be comparable in a way to the effect of the recruitment phenomenon when there is, in addition to general distortion, a high preponderance of loud vowels, whereas the consonants may remain disproportionately weak near the auditory threshold.

SUMMARY.

A short review of papers on per-stimulatory adaptation is given, and some factors affecting binaural simultaneous loudness balance judgment are pointed out. Results are reported in a group of normal subjects, in which the adaptation loss was measured at 60 and 80 db sensation level at 4000, 2000, 1000 and 500 cps. The loudness level loss, measured after three minutes continuous stimulation, was on an average less than 10 db.

In 18 cases of conductive deafness and in nine ears with non-recruiting perceptive deafness, the adaptation loss was comparable to normal values. In recruiting perceptive deafness (51 ears) an abnormally large adaptation loss (30 db or more) was found in 14 per cent. It is concluded that abnormal loudness adaptation is not related to poor speech discrimination, and its occurrence is not so frequent as assumed earlier. When it does occur it seems, however, to be associated with recruiting perceptive deafness.

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DIAGNOSIS AND TREATMENT OF PAROTID CALCULI.

JAMES R. BLAIR, M.D.,

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Parotid calculi are uncommon, occurring more often in men than in women, and rarely attaining the size of calculi found in Wharton's duct. In contrast to the latter they are not often palpable.

Due to local tissue reaction and to overlying structures, parotid stones are as a rule difficult to visualize by ordinary X-ray technique. Uric acid stones, too, since they are not radiopaque, pose a singular diagnostic problem which can be solved only by sialography.

Parotid calculi most often lodge in the masseteric portion of Stenson's duct, at or near the junction of the main and accessory ducts. This fortuitous circumstance makes some of these calculi amenable to conservative treatment, by extraction or lithotrite, thus obviating the unsightly scar resulting from external surgery.

The history of parotid calculi is characteristic. Intermittent, painful pre-auricular swelling aggravated by highly seasoned foods and relieved by a gush of foul, purulent or inspissated saliva, is pathognomonic of ball-valve parotid duct obstruction.

In contrast to infectious parotitis, the glandular swelling from the stone is not always symmetrical; in fact, only certain portions of the gland, especially the accessory lobe, may be enlarged, thus giving rise to the diagnosis of tumor formation. Added evidence may sometimes be gained by probing the duct.

Sialography has proven to be a valuable aid in visualizing and localizing non-opaque uric acid stones. In addition, sialography demonstrates accurately the amount of damage which inevitably results to the gland parenchyma from back pressure.

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The conservative treatment of parotid calculi by direct intraoral extraction, or by lithotrite, is not mentioned in the literature. This method has been successfully employed on a number of occasions. If the stone is located in the intraoral portion of the duct, or even in the anterior portion of the masseter segment, the problem of removal is comparatively simple. The duct is first widely dilated by means of No. 5. ureteral catheters left *in situ* for several weeks. With a small Hartman forceps the stone can then be directly extracted, or, if necessary crushed and extracted piecemeal.

Radical treatment consists of exposing Stenson's duct by a horizontal incision to guard against injury to the nerve. After extracting the stone the duct is repaired by fine silk sutures, and a catheter is left *in situ* for as long a time as seems feasible. By passing the end of the catheter through the eye of a medium-sized skin clip and anchoring this to the buccal mucosa it causes no discomfort.

CASE REPORTS.

Case 1. Mrs. M., an elderly woman with a long history of bilateral, intermittent swellings of the parotid. Examination shows a free flow of pus from both parotid ducts. Patient refused surgery.

Fig. 1. Fig. 2.

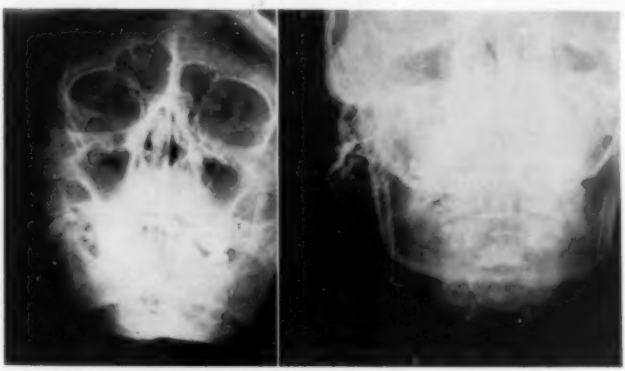

Fig. 1. Showing size and location of parotid stone.

Fig. 2. Sialogram showing destruction of gland parenchyma.

Case 2. J. P. 28-year-old female. Intermittent painful right parotid swelling for 2 years. Recurrent bouts of infection controlled by antibiotics. X-rays failed to show stone. Uric acid stone visualized by sialogram.

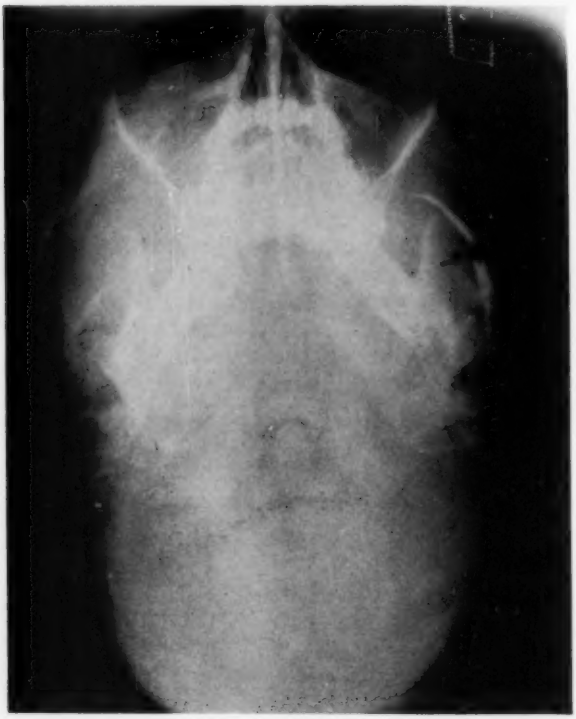

Fig. 3. Sialogram of uric acid stone.

Treatment: Duct dilated by means of catheters and stone removed intraorally with Hartman forceps.

Case 3. Male 51 years of age. History of painful parotid swelling on the right for several years.

Fig. 4. X-ray showing catheter in situ.

Treatment: Stone removed by external incision.

Case 4. 36-year-old male, whose complaint was a continuous discharge of saliva from a wound in the right cheek, which followed an operation for "tumor" several weeks previous. Examination shows a vertical scar in the center of which is an evident salivary fistula.

Fig. 5. Post-operative. Fistula healed.

Operation: Horizontal incision. Stone which was situated at the junction of the main and accessory ducts removed. Fistula repaired. Indwelling catheter. Healing by first intention.

Comment: Pathological report of "tumor" originally removed was "Normal Parotid."

Case 5. 32-year-old male. History of intermittent painful swelling of the left parotid for five years. Operation at angle of the jaw for "tumor" five years previous. Second operation on duct two years ago, which failed to reveal stone.

Fig. 6. Preoperative, showing scar of previous operation on duct and characteristic enlargement of accessory gland.

Operation: Calculus found at the junction of the main and accessory ducts. Calculus removed and stenotic portion of duct excised. Catheter inserted.

CONCLUSIONS.

Parotid calculi are difficult to visualize by ordinary X-ray technique.

Uric acid calculi may readily be visualized by sialography.

Parotid calculi may simulate tumors, especially with enlargement of the accessory lobe when the stone is located at the junction of the main and accessory ducts.

Calculi situated in the intraoral or anterior portion of the masseter segment may sometimes be removed intraorally after wide dilatation of the duct by means of catheters.

Catheters can be worn indefinitely, if fixed to the buccal mucosa by means of skin clips.

227 Sixteenth Street.

MEDICAL NEWS.

Summit, New Jersey, Sept. 8—MEDICAL NEWS, the first newspaper exclusively for the medical profession, will begin publication on September 12, it was announced today by T. F. Davies Haines, president of CIBA Pharmaceutical Products Inc. of Summit, New Jersey. CIBA will distribute the eight-page tabloid-size newspaper free of charge to physicians every other week.

Published for CIBA by Medical News Inc., the new publication will provide the busy physician with brief and authoritative news and feature articles on current medical developments here and abroad. The editorial content of the newspaper will be determined independently of CIBA'S interest as a pharmaceutical manufacturer.

Blake Cabot will serve as executive editor of MEDICAL NEWS. A graduate of Harvard University, Mr. Cabot is the author of *The Motion of the Heart,* a book on cardiovascular research. He was previously director of publications for the Greater New York Fund and has performed public relations work for the American Heart Association. During the past 15 years, Mr. Cabot has written many articles on medical research and public health.

"Reports from special correspondents in the United States, Europe, Latin America and Asia will appear regularly in MEDICAL NEWS," states Mr. Cabot. "These reports," he adds, "will cover clinical Medicine, research, medical education, public health, medical economics and legislation directly affecting the physician."

Accounts of major medical and scientific meetings, now published under the title of CIBA REPORTS, will be incorporated into MEDICAL NEWS. Other regular features will include a European news letter, a review of recently published books on medicine and health, profiles of outstanding physicians and medical scientists, news from medical institutions and medical economics.

MEDICAL NEWS is the latest service provided by CIBA for the medical profession. The CIBA CLINICAL SYMPOSIA, a magazine containing original scientific papers by leading medical professors and practitioners with illustrations by the well-known medical artist, Frank Netter, M.D., is also

distributed free of charge to practicing physicians in the United States. *The CIBA Collection of Medical Illustrations*, published in three volumes, has been made available to physicians at cost. CIBA is also sponsoring a half-hour documentary television program, "Medical Horizons," which will be presented for 26 weeks on the ABC-TV network beginning September 12.

II INTERNATIONAL CONGRESS ON ALLERGOLOGY.

The II International Congress on Allergology will be held in Rio de Janeiro, November 6-13, 1955. The most outstanding authorities on the matter will be present at this meeting, with a view to submitting and considering the following official subjects: Histamin and Mechanism of the Allergological Reactions—(histamin, its metabolism and its rôle on the allergology; histamin releasers. Drug Allergology, including the antibiotics). Immunology and Allergology (chemical compositions of the antigenes and antibodies; immunological quantitative technics applied to allergology). Asthma (physiopathology, death through asthma; climatic asthma). Dermatological Allergology (including industrial and contact dermatitis). Allergology and Verminosis. Allergology and Leprosy. Allergology and Tuberculosis. Allergology and Hormones.

Prior to the opening of the Congress, on November 3-5, an international and intensive course on allergology will be held, in which about thirty famous scientists will participate. The Congressists, by addressing themselves to the Congress Secretary-General, will be considered as registered in this course. All other interested parties (physicians or medical students) are requested to register their names, in due time, at the General Secretariat.

Registration fees: U.S. $15.00. Accompaniers: U.S. $6.00 each.

For further information and registration, please apply to:
Secretaria Geral do
II CONGRESSO INTERNACIONAL DE ALERGIA
Avenida Rio Branco 277, 7° andar—grupo 705
Rio de Janeiro - Brasil.

COURSE IN RECONSTRUCTIVE SURGERY OF THE NASAL SEPTUM AND EXTERNAL PYRAMID.

Under the joint sponsorship of the Departments of Oto-Laryngology of the L.S.U. School of Medicine and Tulane School of Medicine a course in Reconstructive Surgery of the Nasal Septum and External Pyramid will be presented in New Orleans March 10-17 (inclusive), 1956.

The course will be given with the cooperation of the American Rhinologic Society and under the direction of Dr. Maurice H. Cottle.

The assisting guest faculty will include the following: Drs. Walter J. Aageson, Fred W. Beck, Max Bornstein, James Chessen, George G. Fischer, Irwin E. Gaynon, Harvey C. Gunderson, Richard B. Hadley, Robert Hansen, Kenneth H. Hinderer, John A. Kirschner, Walter E. Loch, Roland M. Loring, Joseph Ogura, Charles J. Puerillo, Ivan W. Philpott, Ralph H. Riggs, Pinckney W. Snelling, Carl B. Sputh, Jr., and Russell I. Williams.

Applicants must be diplomates of the American Board of Oto-Laryngology. Class limited. Apply to: Dr. Clifford G. Grulee, Jr., Director of Graduate Medicine, School of Medicine, Tulane University, 1430 Tulane Avenue, New Orleans 12, Louisiana.

BRONCHOESOPHAGOLOGY COURSE

The next Bronchoesophagology Course to be given by the University of Illinois College of Medicine is scheduled for the period October 24 to November 5, 1955, under the direction of Dr. Paul H. Holinger.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

NATIONAL SOCIETY MEETINGS.

Schedule of Meetings for 1956:

Eastern Section, to be held at the Statler Hotel, Boston, Mass., January 13.

Middle Section, to be held at the Netherlands Plaza, Cincinnati, Ohio, January 16.

Western Section, to be held at the County Medical Society Bldg., San Francisco, Calif., January 21.

Southern Section, to be held at the Shamrock, Houston, Texas, January 27-28.

American Board of Otolaryngology, to be held at the Sheraton-Mt. Royal, Montreal, Canada, May 6-11.

American Otological Society, Inc., to be held at the Seigniory Club, Montreal, Canada, May 11-12.

American Laryngological Association to be held at the Seigniory Club, Montreal, Canada, May 13-14.

American Broncho-Esophagological Association, to be held at the Sheraton-Mt. Royal, Montreal, Canada, May 15-16, (afternoons).

The American Laryngological, Rhinological and Otological Society, Inc., will hold its Annual Meeting at the Sheraton-Mt. Royal, Montreal, Canada, May 15-16-17 (mornings only).

Please make early plans to attend the 1956 Spring Meetings in Canada. Both the Seigniory Club and Montreal present most attractive features for you and your family. More information about the places will be told to you.

Reservations at the Sheraton-Mt. Royal Hotel should be made early by addressing the Reservation Supervisor, 1455 Peel Street, Montreal, P. Q., Canada.

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